A CORRELATIVE STUDY ON SERUM LIPID PROFILE AND OSTEOGENIC MINERAL STATUS IN OSTEOPOROSIS

Deepika Lahon[1], Munin Borgohain[2], Barnali B. Thakur[3]

[1]Associate Professor, Department of Biochemistry, Gauhati Medical College & Hospital, Guwahati, Assam.
[2]Associate Professor, Department of Orthopaedics, Fakhruddin Ali Ahmed Medical College & Hospital, Jatigaon, Barpeta, Assam.
[3]Assistant Professor, Department of Biochemistry, Gauhati Medical College & Hospital, Guwahati, Assam.

ABSTRACT

BACKGROUND

Osteoporosis is a condition where the structural and functional integrity of the bones are altered to an extent where the bone material is not sufficiently completed enough to perform its basic function of support, load bearing and component of metabolic pool of osteogenic minerals.

METHODS

The study was conducted in a group of 50 women (25 cases and 25 controls) irrespective of age from different socio-economic status. As per plan of study, the target population was divided into two main groups. The first group was defined as the control group and the other group as the experimental or test group who were admitted in orthopaedic unit of Gauhati Medical College and Hospital with different type of clinical manifestations of osteoporotic disease, the diagnosis of which were made on the basis of x-ray findings, provided the clinical examination and laboratory procedures are in agreement.

RESULTS

In our study, the mean fasting serum TG, HDL and VLDL values are significantly high (P<0.01) and the LDL value is significantly low in the women with osteoporosis in relation to normal control group. Total cholesterol values in osteoporotic group is apparently 6.07% lower than the mean total cholesterol in the normal control group. Osteogenic minerals represented by calcium, phosphate and magnesium show significant elevation (P<0.01) of mean serum concentration in the osteoporotic group than the normal control group.

CONCLUSION

Observed changes and relationship between the lipid profile and mineral status in serum in osteoporosis are indicative of an ongoing rearranging process in an altered metabolism, which is primarily aimed at diminishing its pace towards the associated complications indicated by attempted lowering of circulating cholesterol and maintenance of serum minerals within normal limits with assistance from a hormonal axis composed of components from metabolic-reproductive hormonal axis responsible for modulating natural aging process.

KEYWORDS

Osteoporosis, Lipid Profile, Osteogenic Minerals, Menopause, Aging.

INTRODUCTION

Osteoporosis is the most common metabolic bone disease in the world. It is the second leading cause of musculoskeletal morbidity in elderly. By definition it is an abnormal reduction in bone tissue mass per unit volume of anatomical bone.[1] causing skeletal weakness, although the ratio of mineral to organic elements is unchanged. Osteoporosis is a chronic and progressive metabolic disorder of bone characterized by micro architectural deterioration of bone tissue resulting in increased fragility.[2] Beginning with menopause, women experience accelerated bone loss for about 5-7 years.[3] There is reduction in the number and size of the trabeculae and in the number of osteoblasts present, which leads to fracture or crushes with minor trauma.[4]

Primary osteoporosis is mainly a disease of the elderly, which may be again of two types:

1. Postmenopausal osteoporosis Type I (10-20 years) of menopause.
2. Senile osteoporosis Type II (Aged over 70 years).
3. Idiopathic osteoporosis, which is uncommon but occur in children and young adults of both sexes with normal gonadal function.

Within these different etiological groups of osteoporosis, the late postmenopausal period and age above 70 years is the most susceptible period of life for osteoporotic changes. The common link between these two vulnerable groups for osteoporosis is the definite change in the overall hormonal status associated with menopause and aging.

Oestrogen deficiency is a significant cause of accelerated bone loss in the perimenopausal state and affects circulating levels of specific cytokines.[5] Postmenopausal women with a family history of fractures will probably encounter future problem.[6] The major clinical manifestations of osteoporosis are bone fractures, which cause chronic pain. The remodelling process takes place on bone surfaces in discrete packets known as basic multicellular units.[7] In normal individuals, peak bone mass is reached at age 25-35 years and thereafter a decrease with age occurs in both sexes.[8]

Bones in a dynamic state serve as a reservoir of calcium.[9] The internal metabolism and homeostasis of calcium and phosphate are controlled by PTH, Calcitonin and Vitamin D, all of which act to maintain concentrations of physiologically active ionized calcium and phosphate.[10] Calcitonin secretion is lower in women than in men and declines with age, which leads to excessive bone resorption in women.[11] Oestrogen deficiency both directly and indirectly decreases the efficiency of intestinal and renal calcium absorption and resorption respectively.[12] In support of most of these views on the relationship among osteoporosis, hormonal state and mineral state, most of the workers on the problem suggest changing oestrogen status with the aging process as one of the primary hormonal component.[13]

Lipid profile level increases with increasing age in women, particularly after menopause and osteoporosis more predominantly occurs in the postmenopausal women. So we want to explore a more clear picture regarding osteoporosis and changes in lipid profile, keeping in mind that changes in hormonal profile is a primary factor for both these conditions.

Objectives of the Study
Objective of the study was: (1) To determine the relationship between changes in lipid profile and osteogenic mineral status in osteoporotic individual and to probe the scope for utilization of the interpreted data in formulation of a more rational management of osteoporosis. (2) To interpret the observed affects in association with available information on the related problem regarding osteoporosis.

MATERIALS AND METHODS
The present study was conducted in a group of 50 female subjects (25 cases and 25 controls) irrespective of age taken randomly from different socioeconomic status. As per plan of study, the target population was divided into two main groups. The first group was defined as the control group and the other group as the experimental group or the test group. Experimental or the test group was composed of 25 female patients suffering from osteoporosis based on clinical diagnosis and investigative procedures. Subjects for the control group were selected randomly among women of the different sectors of the society belonging to different occupation and socioeconomic status, co-operating voluntarily. Control group consists of 25 female subjects.

A careful screening was done in selecting the subjects, so that the subjects having any history of pathological fracture, hypertension, diabetes mellitus, cardiac disease, peripheral vascular disease, hepatobiliary disease, renal disorders either in the past or present was not included in this group. Cases taken in the present study for the experimental group were amongst those who were admitted in orthopaedic unit of Gauhati Medical College and Hospital from February 2015 to May 2015 with different types of clinical manifestations of osteoporotic disease, the diagnosis of which were made on the basis of x-ray findings, clinical examinations and laboratory procedures.

Methods of Evaluations
All the biochemical estimations were done by using colorimetric principle in a computer assisted semiautomatic BOEHRINGER 4020 photometer.

The serum was separated and centrifuged for 3 min. at 3000 rpm in a clinical centrifuge machine.

Sample Analysis
Serum triglyceride was estimated by the reagent kits of Dr Reddy's based on glycerol-3-phosphate oxidase peroxidase method developed by Fossati and Prenicipe. Serum cholesterol was determined by reagent kit of Dr Reddy's dependent on enzymatic method with endpoint suitable for colorimetric estimation. HDL cholesterol was determined by the reagent kit based on the cholesterol oxidase and precipitation method. The "Friedewald" formula was utilized to calculate out the serum VLDL from the estimated serum triglyceride level as follows.

VLDL-Cholesterol in mg/dL=TG/5. For determination of serum LDL cholesterol, values of serum total cholesterol, HDL cholesterol and serum triglyceride levels were estimated again by utilizing "Friedewald's" formula, serum LDL cholesterol was calculated.

LDL-Cholesterol in mg/dL=Total cholesterol (TC)–(HDL+VLDL) cholesterol.

Serum inorganic phosphate was estimated by Delsal and Manhouri method (1958), which is a modified method introduced by Gomori in 1942. Protein free filtrate is treated with ammonium molybdate, which reacts with inorganic phosphate to form phosphomolybdenum compound. The hexavalent molybdenum is reduced by means of methyl aminophenol sulphate to give a blue coloured compound, which is estimated colorimetrically. Serum magnesium was estimated by colorimetric method using titan yellow. Serum calcium was determined by the reagent kit of Zydus based on Cresolphthalein complexone method, initially described by Anderegg et al. Serum urea and creatinine were measured by using commercially available assay kits by modified Berthelot method and modified Jaffe's Kinetic method respectively. Serum glucose was estimated by the reagent kit of Dr. Reddy's dependent on glucose oxidase/peroxidase method.

Ethics
The study was approved by Ethics Committee of Gauhati Medical College (GMC).

Statistical Analysis
The results of all the biochemical estimations were statistically analysed and compared between different groups of the study by applying student's "t" test to evaluate the changes of serum lipid profile and minerals (Ca, Po_4, Mg.) in osteoporosis along with few correlation studies depending on preliminary results obtained within some relevant groups.

RESULTS
The details of the results and observations are as follows:
Age
In the normal control group (25 female subjects), the age varied within the range of 20-70 years with a mean age of 34.6±2.50 and a median age of 32 years. Maximum number of subjects are in the class interval of 20-40 years with a relative frequency of 0.632.

In the diseased group, comprising of 25 female subjects the age varied within the range of 40-70 years with a mean age of 61.6±1.58 and a median age of 60 years.

Maximum number of subjects are in the class interval of 41-60 years with a relative frequency of 0.560.

Sex

In the normal control group there are 25 healthy female subjects and in the diseased group there are 25 female subjects suffering from osteoporosis.

Serum Total Cholesterol

In the normal group, the total cholesterol values ranges from 103 to 216mg/dL. The mean value is 167.96±6.44mg/dL and the median value is 176mg/dL. The maximum number of subjects in normal groups have cholesterol activity in the class interval 151-200mg/dL with relative frequency of occurrence 0.597. The minimum number of subjects in this group have total cholesterol in the interval of 101-150mg/dL with relative frequency of occurrence 0.033.

In the patient group, the total cholesterol values ranges from 82-272mg/dL: the mean value is 157.76±8.54mg/dL and the median value is 155mg/dL. The maximum number of subjects in this group have total cholesterol activity in the class interval of 151-200mg/dL with relative frequency of occurrence 0.522. The minimum number of subjects in this group has total cholesterol in the interval of 51-100mg/dL with relative frequency of occurrence 0.042.

Serum Triglyceride

In the normal control group, the serum triglyceride values ranges from 52-180mg/dL. The mean value is 127.32+6.89mg/dL and the median value is 133mg/dL. The maximum number of subjects in this group have serum TG activity in the class interval of 101-150mg/dL with relative frequency of occurrence 0.542, whereas in the patient group mean value is 156.2+6.88 and the median value is 153mg/dL, maximum number of subjects have serum TG activity in the class interval of 151-200mg/dL with relative frequency of occurrence 0.494. So triglyceride values are significantly high in the osteoporotic group than normal control group.

Serum HDL Cholesterol

In the normal control group, the HDL cholesterol values ranges from 25-44mg/dL with mean value of 35.6+/-1.00mg/dL. Maximum number of subjects in this group have serum HDL cholesterol activity in the class interval of 31-40mg/dL with relative frequency of occurrence 0.059. On the other hand in the patient group serum HDL values ranges from 13-62mg/dL, the mean value is 43.16±2.27mg/dL. The maximum number of subjects in patient's group have serum HDL cholesterol activity in the class interval of 31-40mg/dL with relative frequency of occurrence 0.332.

Serum VLDL cholesterol: In the normal control group, the serum VLDL cholesterol values ranges from 10.4±35.8mg/dL, the mean value is 25.39±1.43mg/dL. Maximum number of subjects in control group have serum VLDL activity in the class interval of 21-30mg/dL with relative frequency of occurrence 0.550. But in the patient group VLDL cholesterol ranges from 18-41mg/dL and the mean value is 30.4+1.41mg/dL, the median value is 30mg/dL and maximum number of subjects in osteoporotic group have serum VLDL cholesterol activity in the class interval of 21-30mg/dL with relative frequency of occurrence 0.41.

Serum LDL cholesterol values in the control group ranges from 61.6 to 152.0mg/dL and the maximum number of subjects in this group have serum LDLc activity in the class interval of 101-150mg/dL with relative frequency of occurrence 0.748. (Mean values 109.06±4.67mg/dL).

In the patient group, serum LDL cholesterol values ranges from 38-113mg/dL and maximum number of subjects in this group have serum LDL cholesterol activity in the class interval of 51-100mg/dL with a relative frequency of occurrence 0.521. Only this parameter is low in patients with osteoporosis than the mean values of normal control. (Mean values of patients 81.44±4.87).

Serum Calcium

In control group values ranges from 8-10mg/dL with mean value 9.16±0.124 and the maximum number of subjects in this group have serum calcium activity in the class interval of 8.1-9mg/dL with relative frequency of occurrence 0.589.

In the patient group values ranges from 8.8-10.3mg/dL with mean value 9.74±0.101mg/dL and maximum number of subjects in this group have serum calcium activity in the class interval of 10.1-11.0mg/dL with relative frequency of occurrence 0.501.

Serum Phosphate: In control group, the values ranges from 1.2-4.2mg/dL with mean value 2.95±0.132 and the maximum number of subjects in this control group have serum phosphate activity in the class interval of 2.8-3.5mg/dL with relative frequency of occurrence 0.515.

In the patient group, values ranges from 2.5-4.2mg/dL with mean value 3.28±0.082mg/dL and maximum number of subjects in this group have serum phosphate activity in the class interval of 2.8-3.5mg/dL with relative frequency of occurrence 0.582.

Serum Magnesium

In normal control group, the serum magnesium values ranges from 1.8-2.5mg/dL with mean value 2.20±0.044 and maximum number of subjects in this group have serum magnesium activity in the class interval of 2-2.3mg/dL with relative frequency of occurrence 0.459.

In patient group, the values ranges from 2.1-3.3mg/dL with mean value 2.49±0.064mg/dL and the maximum number of subjects in this group have serum magnesium activity in the class interval of 2-2.3mg/dL with relative frequency of occurrence 0.323.

Serum glucose: In control group, values ranges from 60-95mg/dL with mean value 79.88±1.49mg/dL and the maximum number of subjects in this group have serum glucose activity in the class interval of 71-80mg/dL with relative frequency of occurrence 0.583.

In the patient group values ranges from 82-128mg/dL with the mean value of 97.36±2.31mg/dL and the maximum number of subjects in this group have serum glucose activity in the class interval of 101-200mg/dL with relative frequency of occurrence 0.446.

Serum Creatinine

In the normal control group, the values ranges from 0.7-1.2mg/dL with the mean value 0.95±0.032 and the maximum number of subjects in this group have serum creatinine activity in the class interval of 0.8-1.0mg/dL with relative frequency of occurrence 0.529.

In the patient group, serum creatinine values ranges from 0.7-1.4mg/dL with mean value 1.02±0.035mg/dL and the maximum number of subjects in this group have serum creatinine activity in the class interval of 0.8-1.0mg/dL with relative frequency of occurrence 0.458.

Serum Urea

In the normal control group, the serum urea values ranges from 22-32mg/dL the mean value is 26.04+0.514mg/dL and the median is 26mg/dL. In the patient group serum urea values ranges from 22-67mg/dL, the mean value is 33.0+1.85 mg/dL and the median value is 32mg/dL.

Present study shows a statistically significant increase in TG, HDL and VLDL in osteoporotic group compared with normal control group (P<0.01) Tab 2, 3, 4.

LDL cholesterol is significantly low in osteoporotic group (Tab 5), Fig. 1(B).

No significant difference is observed between mean serum total cholesterol values in two groups (Tab 1), Fig. 1(C).

The group of osteogenic minerals represented by Ca2+, P3-and Mg2+show significant elevation (P<0.01) of mean serum concentrations in the osteoporotic group than the corresponding mean values of the control group (Tab 6, 7, 8), Fig. 1(D).

The mean fasting serum concentrations of glucose in the osteoporotic group show very highly significant increase in comparison to the normal control group (Tab 9).

From the foregoing observations and discussions on the present study, it may be summarily infarct that the osteoporotic group is basically neither frankly hyperlipidemic nor osteogenic dismineralostatic in terms of fasting serum concentrations with respect to normal reference intervals in a population without any clinical osteoporotic evidence. It may be reemphasized again that all the significant changes observed in the osteoporotic group are well within the normal reference interval and the significance of deviation is only with respect to the differences in the mean values with the normal control of the present study. Under the prevailing situation in the present study the lipid profile of the osteoporotic group may be redesignated as basically targeted to minimize cholesterol transport to the periphery and maximize cholesterol scavenging system from the periphery as indicated by lowered circulating total cholesterol and LDL-c with elevated circulating HDLc. In the osteoporotic group, the osteogenic minerals are significantly elevated within the normal reference interval indicating an enhanced rate of mineral turnover between bone and blood, but within the normal limits of renal functions.

The mean serum urea in the osteoporotic group is elevated with very high significance (P<0.001) than normal control. But the serum urea levels in both the groups are well within the clinically normal reference interval (Tab 10).

The mean serum creatinine between the control and osteoporotic group does not show any significant differences (P>0.01) Tab 11.

DISCUSSION

Osteoporosis is a chronic and progressive metabolic disorder of bone, characterized by microarchitectural deterioration of bone tissue resulting in increased fragility. In normal bone, bone formation and bone resorption are closely coupled.

Osteoporosis is unfortunately common, affecting over 20 million people in the United States alone.[14]

Our study was carried out in the Department of Orthopaedics, Gauhati Medical College and Hospital, Guwahati, Assam. The distribution pattern of the age in both the groups shows to balance of the condition as per previously reported age groups as one of the etiological factor of osteoporosis.[15] In the normal control group the highest frequency is observed between the age group 20-40 years, which is explained by the fact that during selection of the control group most of the volunteers are within this group with the natural reluctance of the older group to co-operate in an experimental procedure. Estimations of serum triglyceride, total cholesterol, HDL, LDL and VLDL cholesterol were performed as the index of lipid metabolism and calcium, phosphate and magnesium were selected as the representatives of osteogenic minerals. Estimation of fasting sugar and urea were performed as general index for carbohydrate and nitrogen metabolism and creatinine as an index for renal function. The mean fasting serum TG, HDL and VLDL values are significantly high (p<0.01) and the LDL value is significantly low in the patients with osteoporosis in relation to the normal control group. Osteogenic minerals represented by calcium, phosphate and magnesium show significant elevation (p<0.01) of mean serum concentration in the osteoporotic group than the normal control group. The mean fasting serum concentration of glucose in the osteoporotic group show very highly significant elevation in relation to normal control group. In both the groups, glucose level within the range of 60-110mg/dL. Urea level in serum of osteoporotic group is also elevated than normal control. Serum creatinine levels in osteoporotic group shows a normal range, which excludes the probability of renal involvement in osteoporosis. The correlative part of present study shows that in the osteoporotic group the correlation between total cholesterol and magnesium is significantly increased (r=+0.7) than that of normal control (r=+0.320). The correlation between phosphate and HDL is also increased along with correlations between calcium and magnesium in the osteoporotic group.

There is a significant relationship of phosphate with HDL and LDL cholesterol, the two opposing cholesterol carriers which are inverse to each other, whereas the relationship of triglyceride and total cholesterol is more significant with magnesium and reciprocal in nature. The primary component of osteogenic minerals, calcium shows an increase in correlation with magnesium, but no change in correlation with phosphate in the osteoporotic group [Fig 2(A)].

Filiz has demonstrated that patients with fractures had lower levels of TC, TG, LDL-C than the patients without fractures[16]. In our study LDL cholesterol was significantly low, but there was significant increase in Triglyceride Level (TG) and no significant difference is observed between the mean serum total cholesterol values in the two groups, as most of the cases were found without fractures after radiological evaluation. However, the total cholesterol values in the osteoporotic group is apparently 6.07% lower than the mean total cholesterol in the normal control group.

From most of the available reports on the previous works on osteoporosis by different workers, it emerges out that osteoporosis specially shown in cases of postmenopausal age group is often associated with lipid metabolism disorders in the form of hyperlipidemia and atherosclerotic

complications.[17] Our study corroborate with the findings of Parhami F, Garfinkel A et al. The reports on the serum mineral status appear to be relatively more ambiguous than that of lipid metabolism studies. But among the ambiguity the commonness is that in osteoporotic situations, although the mineral profile of bone is much altered towards the loss of mechanical strength, the serum mineral profile is not proportionately deviated specially shown in situations of age related osteoporotic changes.[18] These two basic informations on the relationship of osteoporosis with serum lipids and minerals lead us to propose that osteoporosis has more probability to be linked up with altered lipid and mineral metabolism with increase in age where the hormonal component may share a common stem. The changing hormonal component of the mineral metabolism is more targeted on metabolism of minerals in bone rather than minerals in circulation. The proposed probability may be one of the explanations of relative absence of drastic alteration in serum mineral profile in presence of gross structural and functional alteration in bone mineral content in osteoporosis.

CONCLUSION

All these observations suggest that no drastic alteration is observed in the serum lipid and mineral profile in osteoporosis. Although minor changes in lipid and mineral metabolism have been observed in osteoporotic subjects, metabolic disorder of bone due to natural aging process like any other living tissues, still seems to be the most plausible explanation for osteoporosis. Larger and more elaborate studies are required to precisely establish the relationship between changes in lipid profile and osteogenic mineral status in osteoporotic individuals. From the available literature on the problems of osteoporosis, dyslipidemias and mineral imbalances, it is felt to be very tough to have a concerted view on the three conditions as most of the works treated them as individual entities.
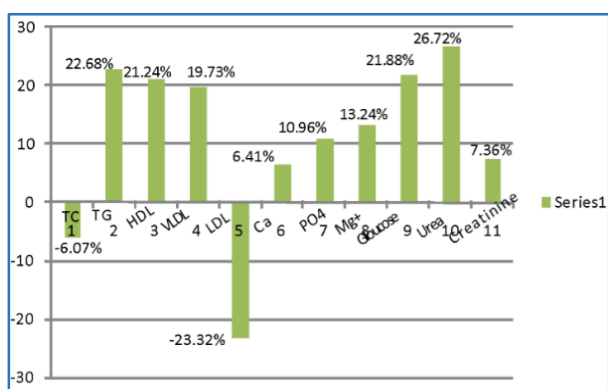
Fig. (I-A)

This figure indicates the mean percent displacement of different parameters in the osteoporotic group from the normal control baseline.

The mean fasting serum TG, HDL and VLDL values are significantly high (p<0.01) and LDL value is significantly low in the patients with osteoporosis in relation to the normal control group. Similarly, osteogenic mineral concentration in serum show significant elevation in osteoporotic group [shown in Fig. (I-B, C, D).

Fig. (I-B)

Fig. (I-C)

Fig. (I-D)

A schematic representation of the relationship between osteogenic mineral and lipid fractions with significant alterations in correlation coefficient in the osteoporotic group.

Fig. 2(A)

During the correlation analysis of the observations of the present study, the relationship between osteogenic mineral axis of Ca^{2+}, Mg^{2+} and PO_4^{3-} is presented against the lipid profile components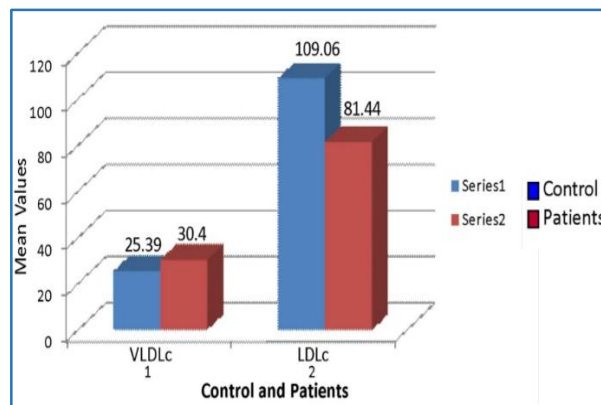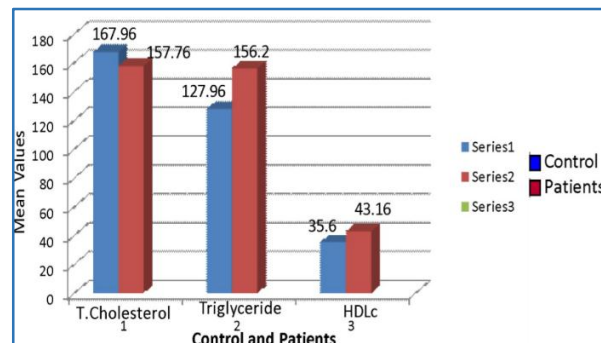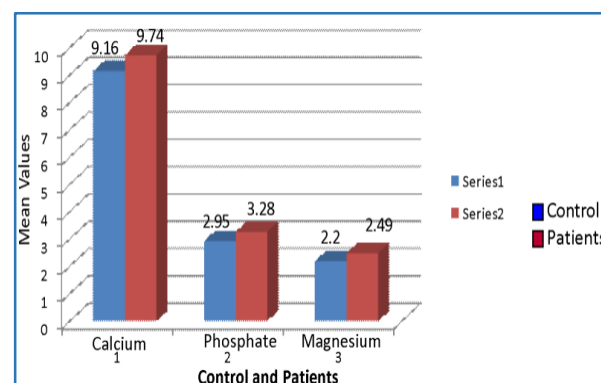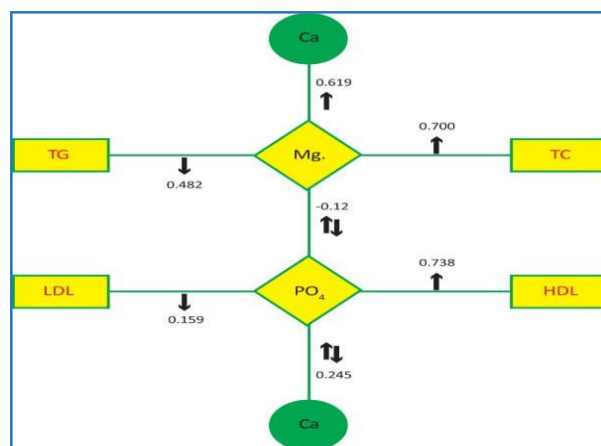 represented by TG, TC, HDL and LDL in the osteoporotic group, it is observed that the relationship of Phosphate with HDL and LDL, the two opposite cholesterol carriers are inversed to each other, whereas the relationship of TG and total cholesterol is more significant with magnesium and also reciprocal is nature. The calcium shows an increase in correlation with magnesium, but no change in correlation with phosphate.

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0-50	0	0	0
51-100	0	0	0
101-150	7	28	0.033
151-200	14	56	0.597
201-250	4	16	0.197

Mode class : 151-200 mg/dl
St Dev±32.23
S.E.M :±6.44
Co-efficient Variant : 19.18

PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0-50	0	0	0
51-100	2	8	0.042
101-150	9	36	0.303
151-200	12	48	0.522
201-250	1	4	0.063
251-300	1	4	0.068

Mode class : 151-200mg/dL
St Dev ± 42.70
S.E.M : ± 8.54
Co-efficient Variant : 27.06

Table 1: Distribution Total Cholesterol in Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0-50	0	0	0
51-100	7	28	0.185
101-150	13	52	0.542
151-200	5	20	0.272
201-250	0	0	0

Mode class : 101-150mg/dL
St Dev ± 34.45
S.E.M : ± 6.89
Co-efficient Variant : 27.05

PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0-50	0	0	0
51-100	1	4	0.023
101-150	10	40	0.325
151-200	11	44	0.494
201-250	3	12	0.155

Mode class : 151-200mg/dL
St Dev ± 34.43
S.E.M : ± 6.88
Co-efficient Variant : 22.0

Table 2: Distribution of Serum Triacylglycerol on Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0-10	0	0	0
11-20	0	0	0
21-30	2	8	0.059
31-40	18	72	0.702
41-50	5	20	0.238

Mode class : 31-40mg/dL
St Dev ± 500
S.E.M : ± 1.00
Co-efficient Variant : 14.0

PATIENT GROUP

C.I.	No. of Pt	%	Rel. Freq.
0-10	0	0	0
11-20	1	4	0.012
21-30	0	0	0
31-40	10	40	0.332
41-50	7	28	0.281
51-60	5	20	0.260
61-70	2	8	0.113
71-80	0	0	00

Mode class : 31-40mg/dL
St Dev ± 11.36
S.E.M : ± 2.27
Co-efficient Variant : 26.3

Table 3: Distribution of Serum HDL Cholesterol in Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0-10	0	0	0
11-20	7	28	0.178
21-20	13	52	0.550
31-40	5	20	0.271
41-50	0	0	0

Mode class : 21-30mg/dL
St Dev ± 7.165
S.E.M : ±1.43
Co-efficient Variant : 28.20

PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0-10	0	0	0
11-20	2	8	0.05
21-30	12	48	0.410
31-40	10	40	0.484
41-50	1	4	0.053

Mode class : 21-30mg/dL
St Dev ± 7.05
S.E.M : ± 1.41
Co-efficient Variant : 23.19

Table 4: Distribution of Serum VLDL Cholesterol in Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0-50	0	0	0
51-100	7	28	0.195
101-150	17	68	0.748
151-200	1	4	0.055
201-250	0	0	0

Mode class : 101-150mg/dL
St Dev ± 23.39
S.E.M : ± 4.67
Co-efficient Variant : 21.44

PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0-50	3	12	0.059
51-100	14	56	0.521
101-150	8	32	0.419
151-200	0	0	0
201-250	0	0	0

Mode class : 51-100mg/dL
St Dev ± 24.37

S.E.M : ± 4.87
Co-efficient Variant : 29.92

Table 5: Distribution of Serum LDL Cholesterol in Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
7.1-8	3	12	0.104
8.1.-9	15	60	0.589
9.1-10	7	28	0.305
10.1-11	0	0	0
11.1.-12	0	0	0

Mode class : 8.1-9mg/dL
St Dev ± 0.624
S.E.M : ± 0.124
Co-efficient Variant : 6.81

PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
7.1-8	0	0	0
8.1-9	2	8	0.072
9.1-10	11	44	0.425
10.1-11	12	48	0.501
11.1-12	0	0	0

Mode class : 10.1-11mg/dL
St Dev ± 0.506
S.E.M : ± 0.101
Co-efficient Variant : 5.20

Table 6: Distribution of Serum Calcium in Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
1.2-1.9	1	4	0.016
2.0-2.7	8	32	0.259
2.8-3.5	12	48	0.515
3.6-4.3	4	16	0.208
4.4.	0	0	0

Mode class : 2.8-35mg/dL
St Dev ± 0.66
S.E.M : ± 0.132
Co-efficient Variant : 22.40

PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
1.2-1.9	0	0	0
2.0-2.7	3	12	0.096
1.8-3.5	15	60	0.582
3.6-4.3	7	28	0.320
4.4-5.1	0	0	0

Mode class : 2.8-3.5mg/dL
St Dev ± 0.414
S.E.M : ± 0.082
Co-efficient Variant : 12.62

Table 7: Distribution of Serum LDL Phosphate in Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
1.6-1.9	3	12	0.099
2.0-2.3	12	48	0.459
2.4-2.7	10	40	0.441
2.8-3.1	0	0	0
3.2-3.5	0	0	0

Mode class : 2-23mg/dL
St Dev ± 0.222
S.E.M : ± 0.044

Co-efficient Variant : 10.1

PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
1.6-1.9	0	0	0
2.0-2.3	11	44	0.323
2.4-2.7	8	32	0.323
2.8-3.1	4	16	0.181
3.2-3.5	2	8	0.104

Mode class : 2-23mg/dL
St Dev ± 0.324
S.E.M : ± 0.064
Co-efficient Variant : 13.0

Table 8: Distribution of Serum LDL Magnesium in Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
60-70	2	8	0.065
71-80	15	60	0.583
81-90	6	24	0.257
91-100	2	8	0.094
101-200	0	0	0

Mode class : 71-80mg/dL
St Dev ± 7.46
S.E.M : ± 1.49
Co-efficient Variant : 9.34

PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
60-70	0	0	0
71-80	0	0	0
81-90	9	36	0.314
91-100	6	24	0.239
101-200	10	40	0.446

Mode class : 100-200mg/dL
St Dev ± 11.56
S.E.M : ± 2.31
Co-efficient Variant : 11.87

Table 9: Distribution of Serum Glucose in Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
21-25	12	48	0.440
26-30	12	48	0.509
31-35	1	4	0.049
36-40	0	0	0
41-45	0	0	0

Mode class : 26-30mg/dL
St Dev ± 2.57
S.E.M : ± 0.514
Co-efficient Variant : 9.88

PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
21-25	5	20	0.139
26-30	6	24	0.206
31-35	5	20	0.201
36-40	8	32	0.372
41-45	1	4	0.081

Mode class : 36-40mg/dL			
St Dev ± 9.29			
S.E.M : ± 1.85			
Co-efficient Variant : 28.1			

Table 10: Distribution of Serum Urea in Normal and Patient Groups

NORMAL GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0.5-0.7	3	12	0.088
0.8-1.0	14	56	0.529
1.1-1.3	8	32	0.382
1.4-1.6	0	0	0
1.7-1.9	0	0	0
Mode class : 0.8-1.0mg/dL			
St Dev ± 0.177			
S.E.M : ± 0.035			
Co-efficient Variant : 17.44			
PATIENT GROUP			
C.I.	No. of Pt	%	Rel. Freq.
0.5-0.7	1	4	0.027
0.8-1.0	13	52	0.458
1.1-1.3	10	40	0.458
1.4-1.6	1	4	0.054
1.7-1.9	0	0	0
Mode class : 0.8-1.0mg/dL			
St Dev ± 0.161			
S.E.M : ± 0.032			
Co-efficient Variant : 16.95			

Table 11: Distribution of Serum Creatinine in Normal and Patient Groups

Parameters	Normal	Patient
Serum Magnesium	P<0.005	P<0.01
Serum Calcium	P<0.0005	P<0.0005
Serum Phosphate	P>0.01	P<0.025
Serum Urea	P<0.005	P<0.01
Serum Total Cholesterol	P>0.1	P>0.01
Serum Creatinine	P>0.05	P>0.05
Serum Triglyceride	P<-.005	P<0.01
Serum HDLC	P<0.005	P<0.01
Serum VLDLC	P<0.01	P>0.005
Serum LDLC	P<0.0005	P<0.01

Table 12: Table showing the P values for both Control and Patient Groups of Study

REFERENCES

1. Solomon L. Bone loss in ageing individuals. Orthopaedics 1978;6:121.
2. Hodsman AB, Drost DJ, Goldenberg J. Reduced vertebral fracture rates in osteoporotic patients responding to fluoride treatment with an increasing vertebral bone mineral density. In: Christian Sen C, overgaard K, eds. Osteoporosis. Copenhagen osteopress APS, 1990;1484-1485.
3. The Merk manual of diagnosis and therapy. Section 5 Musculoskeletal and connective tissue disorders. chapter 57, osteoporosis. pg 450.
4. Mercer's orthopaedic surgery by Robert Duthie and George Bentley, 9th edn:pg 312.
5. Orimo H, Shiraki M, Inoue S. Oestrogen and bone. Osteoporosis Int 1993;3(I):153-156.
6. Brown EM. Fluoride and the therapy of osteoporosis. J Clin Endocrinol Metab 1989;69(5):929-931.
7. Parfitt AM. Bone remodelling and bone loss; understanding the pathophysiology of osteoporosis. Clin Obstet Gynecol 1987;30(4):789-811.
8. Melsen B, Melsen F, Mosekilde L, et al. Histomorphometric analysis of normal bone from the iliac crest. Acta path Micr Scand Sect A 1978;86:70-81.
9. Satynarayana U, Chakrapani U. Textbook of Biochemistry. 4th edition:pg 404.
10. Harrison HE, Harrison HC. The interaction of vitamin D and PTH on Ca, P and Mg homeostasis in the rat. Metabolism 1964;13(10):952-58.
11. Macleod John ed, Davidson's principles and practice of Medicine. 14th ed:pp 70.
12. Mazzuoli GF, D' Erasmo E, Minisola S, et al. Pathogenic aspects of involutional osteoporosis. Clin Rheumatol 1989;8(2):22-29.
13. Osteoporosis: Diagnosis and treatment. David J Sartosis. Copy right by Marcel Dekker Inc., New York, Indian reprint 2000;pg 6-9.
14. National institutes of health consensus development conference statement on osteoporosis. JAMA 1984;252:799-802.
15. Apley's System of orthopaedics and fractures. A graham Apley, Lousis Solomon. 8th ed:pp 185.
16. Filiz Sivas, Ebru Alemdaroglu, Eda Elverici, et al. Serum lipid profile: its relationship with osteoporotic vertebrae fractures and bone mineral density in Turkish post-menopausal women. Rheumatology International 2009;29(8):885-890.
17. Parhami F, Garfinkel A, Demer LL. Arterioscler Thromb Vasc Biol. American Heart Association 2000;20(11):2346-8.
18. Samual L Turek. Orthopaedics principles and their application. JB Lippincott, Philadelphia, Chapter 9, 4th Ed:pp 241.