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# ASSOCIATION OF VITAMIN D3 DEFICIENCY IN NONALCOHOLIC LIVER DISEASE

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**ABSTRACT**

The crucial secosteroid hormone Vitamin D has an influence on the homeostasis of calcium, however, in the recent years, there have been growing idea that this vitamin also contributes to cell differentiations and proliferations, and it acts as an anti-inflammatory and immunomodulatory factor. Deficiency of Vitamin D is commonly stated in several causes of chronic hepatic diseases and is related to evolution and development of the non-alcoholic fatty liver diseases (NAFLDs) and in infection with chronic hepatitis C (CHC). The roles of Vit. D in the pathogenicity of CHC and NAFLD have not entirely recognized, but it appears that, Vit. D's contribution to the regulations and activations of the adaptive and innate immune responses as well as its anti-proliferative effects can clarify its significance in such hepatic diseases. Available studies indicated performing routine testing for hypovitaminosis D in hepatic disease patients. For this study, we did in methodology some tests that associated with NAFLD to identify effecting biomarker such as Liver Function tests and Lipid profile tests and Diabetes profile test. Further prospective studies demonstrating the impact of vitamin D replacement in NAFLD is required. Aim of study to identify effecting of deficiency Vit D3 for nonalcoholic liver disease.

**KEYWORD:** Vitamin D, NAFLD, Vit D3, Liver Function tests, hypovitaminosis D, hepatitis C (CHC).

## INTRODUCTION

The fat-soluble Vitamin D (also known as "calciferol") is naturally found in some foods, added to other foods and exist as food supplements. This endogenous production of this vitamin occurs when the skin is exposed to the sunlight's ultraviolet rays causing vitamin D formation (NIH, 2022). Several health benefits are offered by Vitamin D3, such as bone and muscle strengthening, immunity boosting, mood improvement, inflammation reduction as well as heart function improvement (Yvelette Stines, 2021). Many hepatic diseases are related to osteoporosis, and the deficiency of vitamin D may strongly exacerbate osteoporosis. Also, several post-transplantation individuals are subject to enhanced bone losses, which may be deteriorated by the lack of vitamin D. Decreased Vit. D levels may also seriously rise some interferon therapy side effects, like muscle ache. Some studies reported that patients with hepatitis C who are vitamin D deficient respond weakly to interferon therapies (Satheesh Nair, 2010). Deficiency of Vit. D was commonly stated in several causes of chronic hepatic disease and was related to evolution and development of the non-alcoholic fatty liver diseases (NAFLDs) and in infection with chronic hepatitis C (CHC). The role of vitamin D in the pathogenesis of NAFLD and CHC is not completely known, but it seems that the involvement of vitamin D in the activation and regulation of both innate and adaptive immune systems and its Anti-proliferative effects might demonstrate its significance in such hepatic disorders (World J Hepatol., 2014). Levels of some proteins and enzymes in the blood are checked by Liver function tests. Higher or lower levels than normal may suggest hepatic disorders. Some frequent liver function tests involve: aspartate transaminase (AST), alanine transaminase (ALT) as well as others used to complete this research (www.mayoclinic.org). The term non-alcoholic fatty liver disease (NAFLD) indicates a group of disorders resulting from fat build-up in the liver. It is commonly observed in obese or overweight individuals. The early-stage NAFLD often causes no harms, but it may cause severe hepatic damages, such as cirrhosis, when it is worsened ned (www.nhs.uk). Most people with NAFLD will not develop any serious problems, These people can treatment with Medicine and liver transplant. (www.nhs.uk). The non-alcoholic fatty liver disease (NAFLD) is a pathological disorder described by accumulation of abnormal triglycerides in the hepatocyte, and sometimes accompanied by necroinflammatory activities with fibrosis

(steatohepatitis) and may develop to hepatic cirhosis. NAFLD is the major commonly observed chronic hepatic disease in the world, that reaches >70% prevalence in patients with type 2 diabetes (T2D), obesity and metabolic syndrome (Chalasani N., Younossi Z. and others, 2012). In these groups, NAFLD may significantly worsen metabolic outcomes and, in the general populations, is recently regarded as one of the independent risk factors for cardiovascular diseases and the most common public health problem.[3,4] Nevertheless, in addition to lifestyle interventions, no recognized treatment for NAFLD has been defined till now (Liyanagedera S., Williams R.P. and others, Pharmacological NAFLD management in adolescents and children, 2017). Vitamin D is the hormone that exerts many advantageous impacts other than its roles in bone homeostasis. The active form of this vitamin was demonstrated to contribute in the immune system modulation, leading to induction of the hepatic anti-inflammatory and anti-fibrogenic patterns (Beilfuss A., Sowa J.P and others, 2015). Nonetheless, the vitamin's protective actions against fibrosis was not effective on the establishment of obvious hepatic cirrhosis (Abramovitch S., Sharvit E. and others, 2015). In addition, vitamin D was suggested as an efficient modulators of insulin sensitivity in many experimental samples, and epidemiological studies showed the presence of a strong relationship between the low levels of circulating vitamin D and obesity, T2D as well as insulin resistance-associated disorders (Bell N.H., and others, 1985)**.**

## 2.1. MATERIAL AND METHOD

This chapter includes the design of the study which includes the administrative arrangements, the study setting, the study sample and selection of sample criteria, the study instruments, pilot study, data collection and analysis, and study limitation.

### 3.2. Liver function tests

- Albumin
- Total proteins. This test includes the total blood protein measurement.
- ALP (alkaline phosphatase), ALT (alanine transaminase), AST (aspartate aminotransferases), which are the enzymes made in the liver.
- Bilirubin, the waste product synthesized in the liver.

**ASPARTATE AMINOTRANSFERASE (AST) testing Procedure**

The user's guide for the analyzer provides all the test parameters and operational procedures

Materials providing AST reagents

-Final reaction mixture's stability

All the determinations are computed automatically by the Beckman Coulter AU analyzer at the same time intervals.

The AST procedure's calibration on AU400/400e & AU600/640/640e depends on theoretical extinction coefficients of

NADH, that have 4960 molar absorptivity at 340/380 nm, while on AU5800/2700/5400/680/480, it depends upon experimentals determinations of molar absorptivity in 340/660 nm.

**Quality Control**

During operation of the Beckman Coulter AU analyzer at least two levels of an appropriate quality control material should be tested a minimum of once a day. the control must be done with every new portion of reagent, and following particular steps of troubleshooting or maintenance shown in the User's guide. Testing of the quality control must be accomplished according to the regulatory requirement and the standard procedures of each lab.

**Results**

For all samples, the results are automatically printed at 37°C in U/L.

**ALANINE AMINOTRANSFERASE (ALT) Test Procedure**

The user's instructions provides all the test parameters and operational procedures

**MATERIALS**

1. ALT Reagents
2. Pipe (one per each 180mL vial).

**Final reaction mixture's stability**

All the determinations are computed automatically by the Beckman's Coulter AU analyzers at the same time intervals. The ALT procedure's calibration on AU400/400e & AU600/640/640e depends on theoretical extinction coefficients of NADH, that have a molar absorptivity in 4960 at 340/380 nm, while on AU5800/2700/5400/680/480 it depends upon experimental determinations of molar absorptivity in 340/660 nm.

**Quality Control**

At least two level of suitable materials of quality controls should be examined once a day during the Beckman's Coulters AU analyzer operation. In addition, the control must be done with every new portion of reagent, and following particular steps of troubleshooting or maintenance shown in the instructions for users. Testing of the quality controls must be accomplished according to the regulatory requirement and the standard procedures of each lab.

**Results**

For all samples, the results are automatically printed at 37°C in U/L.

**TOTAL BILIRUBIN Test PROCEDURE**

The user's instruction provides all the test parameters and operational procedures

Materials providing bilirubin reagents

- Materials Required: Calibrator (Catalouge No. DR0070)
- Ultimate reactions mixture's stability.

Beckman's Coulter AU analyzers automatically compute all the determinations at the same time.

**Calibrations**

Calibrations must be performed every 30 day. Chemistry calibrators (Cat DR0070) is used to calibrate the total bilirubin procedure), which matches with the National institute of standard & technology (NIST), Standard reference materials (SRM) 916a. in case of existence of these conditions, re-calibrations of the tests are needed.

1. Changing of reagent lot's number or observance of any shift in the control's value.
2. Performance of the major preventative's
3. maintenance on analyzers.
4. Replacement of critical parts.

**Quality Control**

During operating the Beckman's Coulter AU analyzers, at least two suitable levels of quality controls materials should be tested with minimally once per day. Also, the control must be done with every new portion of reagents, and following particular steps of troubleshooting or maintenance shown in the User instruction. Testing of the quality controls must be accomplished according to the regulatory requirement and the standard procedures of each lab.

**RESULTS**

For all samples, the results are automatically printed at 37°C in mg/L. For SI unit (µmol/L), the results should be multiplied by 17.1.


**Figure (3-1): Beckman Coulter / Olympus AU 480 Chemistry Analyzer.**

**3.1 Diabetes tests ( FBS, HbA1C)**

A simple blood sugar test procedure allows measuring the glucose in the blood, and this test may be requested by a doctor when he suspects that the blood sugar is abnormal. Diabetic people can highly make use of the test for monitoring levels of blood glucose.

**3.1.1 Fasting blood sugar (FBS)**

FBS is measured before people have eaten. Normal FBS range is (70-100) mg/dl. A result between (100-126) mg/dl is regarded as impaired fasting sugar or pre-diabetes condition. The diagnosis of diabetes is usually established if the level of FBS is ≥ 126 mg/dl. Simple complications may occur e.g. puncture site infection, bleedings, or difficulty to find a vein.

**Fasting test preparation**

To do the fasting blood sugar test, the patient can drink or eat nothing except water for 8 hrs. prior to the test. A scheduled fasting glucose tests must be prepared first in the morning, so the day time fasting is not needed. Fasting before a blood glucose test is important because it provides more precise result making it easier to interpretation by doctors.

**3.1.2 HbA1c**

The amounts of blood glucose bound to hemoglobin A over the last three months is measured by the HbA1c test. It is the three month average because it indicates the period of a red blood cell life span. Hemoglobin is the component of the red blood cell which carries oxygen from the lung to other body parts.

What happens during an HbA1c test?

Using a small needle, the blood sample is taken from the arm vein by health care individual, and a small quantity of blood is obtained and put in test tubes. The patient might feel small pain on needle entrance or going out the vein. The process often takes 5 minutes or less.

You don't need any special preparations for an HbA1c test. Results of HbA1c are recorded in a percentage. The typical result is shown below.

- Normal HbA1c is less than (5.7%).
- Prediabetic HbA1c = (5.7% - 6.4%)
- Diabetic: HbA1c = (≥ 6.5%).

**4.2 Lipid profile tests (TG, Cholesterol, LDL, HDL, VLDL and albumin)**

**High density lipoprotein (HDL)**

HDL is a good cholesterol since it helps in removal of cholesterol from tissues and removal of excessive cholesterol from depositing in the arteries, thereby protecting from cardiac diseases. The low levels of serum HDL-cholesterol is related to high risks of CHD. When the HDL levels decrease from 30-40 mg/dl, there will be a marked increase for coronary risks. A low HDL-cholesterol level is supposed to be < 35 mgs/dls, and high HDL >60 mgs/dls. The value of HDL-cholesterol is also applied to calculate LDL cholesterol.

**Low density lipoprotein (LDL)**

LDL is a bad cholesterol since it functions against the functions of HDL leading to cholesterol deposition in the arteries thereby increasing risks of cardiac disease. The majority of circulating cholesterol exist in three main lipoprotein fraction: HDL, LDL and very low density lipoprotein (VLDL).

**Total cholesterol = (VLDL- cholesterol) + (LDL-cholesterol) + (HDL-cholesterol)**

The level of LDL-cholesterol is calculated from measuring the value of total cholesterols, triglyceride as well as HDL- cholesterol in accordance with the relationships.

**(LDL-cholesterol) = (Total cholesterol) – (HDL-cholesterol) – (Triglyceride) / 5**

In humans, the majority of circulating cholesterol is carried by LDL, and when it is increased it will be involved in the initiation of coronary atherosclerosis. The level of LDL-cholesterol is measured for assessing the CHD risks and following up the progress of individuals who are treated to decrease the levels of LDL-cholesterol. Appropriate LDL-cholesterol levels are < 130 mgs/dls in the adult and 110 mgs/dls in the child.

Chylomicron: It transports triglycerides from the intestine (exogenous die try TG)

VLDL (15% cholesterol): It transports triglycerides from liver to tissues (endogenous triglycerides) LDL (60% cholesterol): it transports cholesterol to tissues

HDL (25% cholesterol): it transports cholesterol from tissues.

**Procedure**

1- The cholesterol mono reagent (MR) and the cholesterol standard (50 mg/dl) were brought to room temperatures.
2- They were pipetted into a labelled tube

| | Blank | Standard | Sample |
|---|---|---|---|
| Standard | | 50 µL | |
| Sample | | | 50 µL |
| Reagent 1 | 1.0 mL | 1.0mL | 1.0mL |

3- They were mixed and let to stand at RT for 10 min.
4- The supernatant's and standard's absorbance (A) is read at 500 nm wave length against blanks.

***Calculation***

$$1-\ C\ sample = \frac{Abs\ sample}{Abs\ standard} \times C\ standard$$

$$= mg/dl$$

$C_{sample}$ = concentration of sample (unknown)

$C_{st.}$ = concentration of standard ( mg/dl)

$Abs_{sample}$ = absorbance of the sample

$Abs_{st.}$ = absorbance of standard

Conversion factor = C mg/dl × 0.0259

= C m mol/L

2- VLDL= TG/5

3- LDL= Total cholesterol- (HDL+ VLDL)

The color is stable for at least 30 minutes protected from light.

**RESULT AND DISCUSSION**

The findings of this study were shown systematically in tables according to the study's objectives.

**4.1 Results**

**Table 4-1: Distribution of the study groups by (gender, age, BMI).**

| SD Cv. | Groups | Study | | Control | | Total No. (%) | *P*-value |
|---|---|---|---|---|---|---|---|
| | | No. | % | No. | % | | |
| Gender | Male | 15 | 50 | 6 | 30 | 21(42) | *P*= 0.01* H S |
| | Female | 15 | 50 | 14 | 70 | 29 (58) | |
| | Total | 30 | 100 | 20 | 100 | | |
| Age Groups (Per years) | 20_29 | 3 | 10 | 6 | 30 | 9(18) | *P*= 0.00* H S |
| | 30_39 | 8 | 27 | 6 | 30 | 14( 28) | |
| | 40_49 | 7 | 23 | 6 | 30 | 13(26) | |
| | 50_59 | 7 | 23 | 0 | 0 | 7(14) | |
| | >59 | 5 | 17 | 2 | 10 | 7(14) | |
| | Total | 30 | 100 | 20 | 100 | 50(100) | |
| | | Study no.=30 | | Control no.=20 | | | |
| BMI | <20 | 0 | 0 | 3 | 15 | | *P*=0.0000* H S |
| | 20-30 | 11 | 37 | 17 | 85 | 28(56) | |
| | >30 | 19 | 63 | 0 | 0 | 19(38) | |
| | Total | 30 | 100 | 20 | 100 | | |

* Highly significant; ** non significant

(**) NS: Non-significant at P>0.05. The test depends on a contingency coefficient testing.

This table shows highly significant correlation between gender, age and BMI at p=0.00, regarding gender female distribution was more predominant 29 (58%) while males were 21(42%) in all study groups, regarding age groups in all study groups the results showed that (30-39) years old were 14(28%) more ages distributed in this study while 28(56%) were the more distributed BMI in this study samples as (20-30) BMI at p-value (0.000) HS.

**Table 4-2: Correlation of BMI with HbA1c, FBS, TSB in all study groups.**

| TESTS | Range | Study (LIVERdz) | | *P*-value |
|---|---|---|---|---|
| | | BMI | | |
| | | 20-30 | >30 | |
| | | No (%) | No (%) | |
| HbA1c % | 4-6% | 7(23%) | 13(43%) | *P*=0.34**NS |
| | > 6 | 5(17%) | 5(17%) | |
| | Total | 12(40%) | 18(60%) | |
| FBS Mg/dl | <100 | 2(7%) | 5(17%) | *P*=0.9**NS |
| | 100-120 | 4(13%) | 7(23%) | |
| | >120 | 4 (13%) | 8 (27%) | |
| | Total | 10(33% | 20(67%) | *P*=0.6**NS |
| TSB Mg/dl | 0.2-1 | 9(30%) | 12(40%) | |
| | > 1 | 3(10%) | 6(20%) | |
| | Total | 12(40%) | 18(60%) | |
| *P*-value | 0.73 **NS | | | |

*(HS) Highly significant; ** (NS) non significant.

This table shows non-significant correlation (p=0.73) of BMI with HbA1c, FBS & TSB in liver diseased patients group as well as patients with BMI >30 showed (43%) more HbA1c level at 4-6% with NS correlation at p=0.34, while patients having FBS >120 showed (27%) with BMI >30 at p=0.9, also patients with TSB ranged (0.2-1) showed (40%) having BMI >30 at p=0.6.

**Table 4-3: Correlation of age with increase BMI, GOT, GPT, ALP and TSB in patients with liver disease.**

| S D Cv. | Study abnormal liver tests *No =30* | | | | | | *Vit. D< 30 (30-100)* | *P*-value |
|---|---|---|---|---|---|---|---|---|
| | Tests | BMI> 30 | GPT> 78 | GOT> 37 | ALP> 164 | TSB> 1 | | |
| | Unit | | I U/ L | I U/ L | I U/L | Mg/dl | nmol/L | |
| | | No% | No% | No% | No% | No% | No% | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Age Groups (Per years) | 20_29 | 1 (3%) | 1 (3%) | 1 (3%) | 0 | 1 (3%) | 2 (7%) | P= 0.008 * H S |
| | 30_39 | 4 (13.5%) | 2 (7%) | 6 (20%) | 2 (7%) | 4 (13.5%) | 2 (7%) | |
| | 40_49 | 6(20%) | 1(3%) | 1(3%) | 1(3%) | 1(3%) | 2(7%) | |
| | 50_59 | 4 (13.5%) | 3 (10 %) | 3 (10%) | 2 (7%) | 3 (10%) | 3(10%) | |
| | >59 | 3 (10%) | 0 | 0 | 0 | 0 | 3(10%) | |
| | Total | 18 (60%) | 7(23%) | 11(37%) | 5(17%) | 9(30%) | 12(41%) | |

* Highly significant (HS); ** non significant (NS).

This table shows highly significant correlation of age with abnormal results of BMI, GOT,GPT,ALP & TSB at p=0.008, according to age groups the age group (40-49) showing increase BMI at (20%) and decrease vitamin D less than 30 were only (7%) with normal level of liver function tests where as another age group (50- 59) showed increase BMI >30 at (13.5%) with increase GPT (10%), GOT (10%), ALP (7%) and TSB (10%) above normal level with 10% of patients showed decrease vitamin D <30 at HS (p=0.008).

**Table 4-4: Correlation of gender & abnormal BMI with increase lipids and albumin in patients with liver disease.**

| Tests Mg/dl | BMI> 30 | | gender | | | | *p*-value |
|---|---|---|---|---|---|---|---|
| | | | Male | | Female | | |
| | No. | % | No. | % | No. | % | |
| S.Cholesterol>200 | 6 | 20 | 4 | 13 | 7 | 23 | *P=0.01 *HS* |
| S.TG >150 | 7 | 23 | 8 | 27 | 6 | 20 | |
| HDL >60 | 4 | 13 | 2 | 7 | 3 | 10 | |
| LDL >100 | 10 | 33 | 7 | 23 | 11 | 37 | |
| VLDL >30 | 7 | 23 | 8 | 27 | 5 | 17 | |
| S.albumin>3.5 | 16 | 53 | 11 | 37 | 13 | 43 | |

* Highly significant (HS); ** non significant (NS).

The results of this table showed HS correlation between distribution of gender and BMI>30 with increase lipid profile tests (TC, TG, HDL, LDL, VLDL), increase cholesterol >200 more in females (23%) than males while TG>150 was more in males (27%) than in females as well as the level of LDL was very high in patients with BMI>30 at (33%), with (23%) in males and (37%) in females, also serum albumin level>3.5 was very high in females (43%) than males (37%).

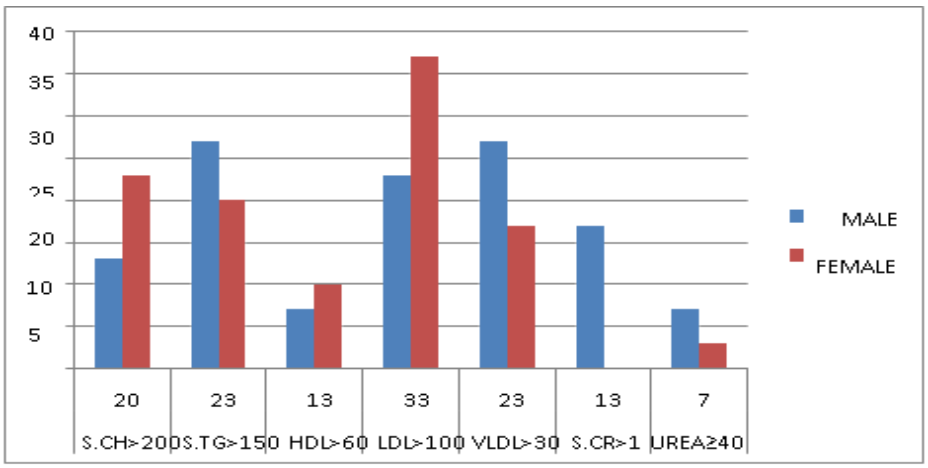


**Figure 4-1: Correlation of BMI with HbA1c, FBS, TSB in all study groups.**

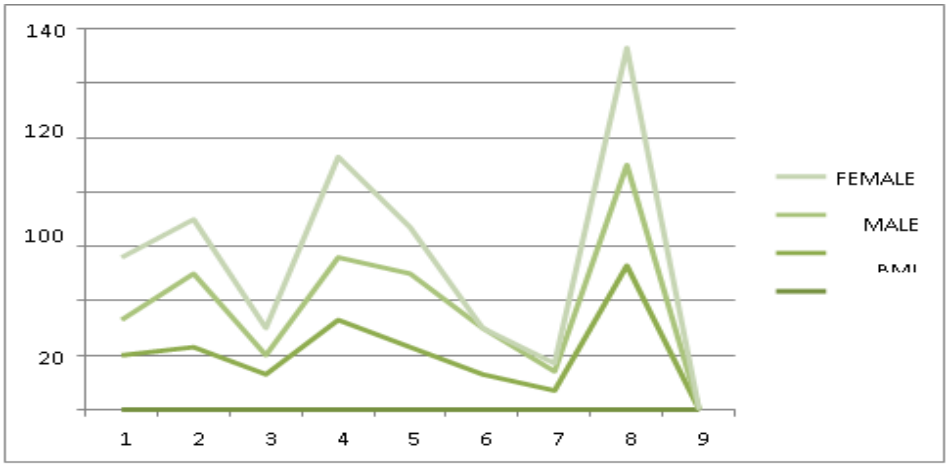


**Figure 4-2: Correlation of gender & abnormal BMI with increase lipids and albumin in patients with.**

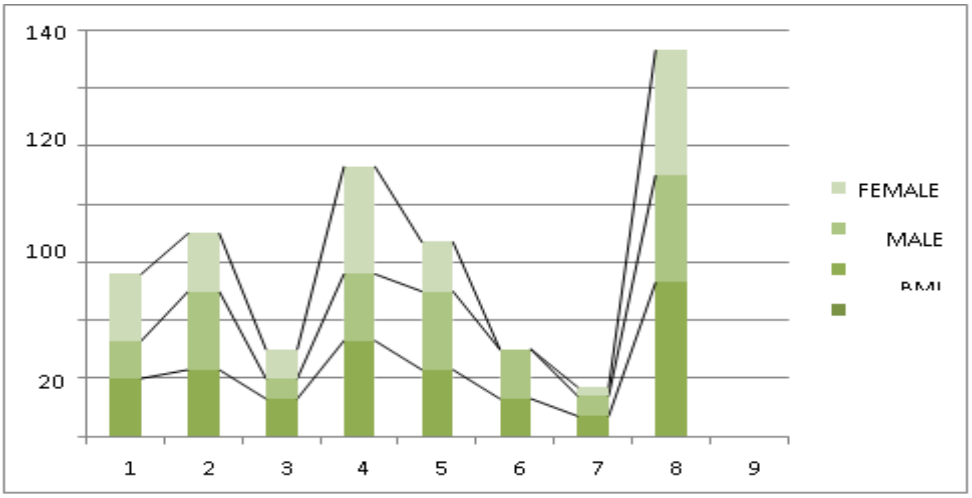


**Figure 4-3: Correlation of gender & abnormal BMI with increase lipids and albumin in patients with.**

## 3. RESULTS AND DISCUSSION

The findings of this study were shown systematically in tables according to the study's objectives as follows: 4.1 Results Table 4-1: Distribution of study groups according to (gender, age, BMI).

### 5.1 CONCLUSION

1. Patients with NAFLD had decreased serum levels of vitamin D, indicating that this vitamin might play an essential role in NAFLD developments. For determination of good therapeutic implications of Vit. D, future studies are needed for prophylaxis or treatments of NAFLD.

2. Low vitamin D is prevalent in chronic liver disease patients. Even patients with mild liver disease are affected, although patients with liver cirrhosis more frequently have serious deficiencies. Decreased levels of Vit. D were seen in chronic liver disease, particularly in hepatic cirrhosis, whereas in NAFLD patients, there are still little data.

3. The pleiotropic impacts of Vit. D suggest an association between the vitamin's deficiency and many chronic diseases, like diabetes, cardiovascular disease, autoimmune and infectious disease, many cancer types as well as chronic hepatic disease.

4. LDL was very high in patients, while FBS & TSB in liver diseased patients.

### 5.2 Recommendation

1. Require studies demonstrating the impact of vitamin D replacement in NAFLD is required.

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