IJMPR, 2026, 10(4), 63-70 ISSN: 2319-5878

INTERNATIONAL JOURNAL OF MODERN PHARMACEUTICAL RESEARCH

www.ijmpronline.com

Research Article

Impact Factor: 7.533

Coden USA: IJMPR3

ASSOCIATION OF VITAMIN D3 DEFICIENCY IN NONALCOHOLIC LIVER DISEASE

Ibtihal Hashim Azeez*

Article Received on: 05/03/2026 **Article Revised on: 25/03/2026** **Article Published on: 04/04/2026**

***Corresponding Author**

Ibtihal Hashim Azeez

https://doi.org10.5281/zenodo.19437317

How to cite this Article: Ibtihal Hashim Azeez* (2026). Association of Vitamin D3 Deficiency in Nonalcoholic Liver Disease. International Journal of Modern Pharmaceutical Research, 10(3), 63–70.

ABSTRACT

Vitamin D is an important secosteroid hormone with known effect on calcium homeostasis, but recently there is increasing recognition that vitamin D also is involved in cell proliferation and differentiation, has immunomodulatory and antiinflammatory properties. Vitamin D deficiency has been frequently reported in many causes of chronic liver disease and has been associated with the development and evolution of non-alcoholic fatty liver disease (NAFLD) and chronic hepatitis C (CHC) virus infection. 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 ant proliferative effect may explain its importance in these liver diseases. Published studies provide evidence for routine screening for hypovitaminosis D in patients with liver disease. 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

Vitamin D (also referred to as “calciferol”) is a fat-soluble vitamin that is naturally present in a few foods, added to others, and available as a dietary supplement. It is also produced endogenously when ultraviolet (UV) rays from sunlight strike the skin and trigger vitamin D synthesis. (NIH, 2022) Vitamin D3 offers many health benefits. It: Strengthens bones and muscles, Boosts immunity, Improves mood, Reduces inflammation and Improves heart function. (Yvelette Stines, 2021) Some liver disease can be associated with osteoporosis, and vitamin D deficiency can potentially exacerbate that. Many post- transplant patients are also prone to accelerated bone loss, which could be worsened by vitamin D deficiency. Dangerously low levels of vitamin D may also increase some side effects of interferon therapy, such as muscle aches. There are some reports of hepatitis C patients with vitamin D deficiency responding poorly to interferon therapy. (Satheesh Nair, 2010) Vitamin D deficiency has been frequently reported in many causes of chronic liver disease and has been associated with the development and evolution of nonalcoholic fatty liver disease (NAFLD) and chronichepatitis C (CHC) virus infection. 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 antiproliferative effect may explain its importance in these liver diseases. (World J Hepatol., 2014) Liver function tests check the levels of certain enzymes and proteins in your blood. Levels that are higher or lower than normal can indicate liver problems. Some common liver function tests include: Alanine transaminase (ALT), Aspartate transaminase (AST) and others used to complete this research (www.mayoclinic.org) Non-alcoholic fatty liver disease (NAFLD) is the term for a range of conditions caused by a build-up of fat in the liver. It's usually seen in people who are overweight or obese. Early-stage NAFLD does not usually cause any harm, but it can lead to serious liver damage, including cirrhosis, if it gets worse.(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). Non-alcoholic fatty liver disease (NAFLD) is a pathological condition characterized by aberrant triglycerides accumulating in the hepatocytes, in some cases accompanied by necro-inflammatory activity and fibrosis (steatohepatitis) and potentially evolving into liver cirrhosis. NAFLD represents the most common chronic hepatopathy worldwide, reaching a prevalence of above 70% 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

population, is now considered an independent risk factor for cardiovascular disease and a major public health issue.[3,4] However, beside lifestyle intervention, no established therapy of NAFLD has been identified yet (Liyanagedera S., Williams R.P. and others The Pharmacological Management of NAFLD in Children and Adolescents. 2017). Vitamin D is a hormone exerting several beneficial effects beyond its role in bone homeostasis; active vitamin D has been shown to modulate the immune system, inducing an anti-inflammatory and anti-fibrogenic pattern in the liver (Beilfuss A., Sowa J.P and others, 2015). However, its protective action against fibrosis was ineffective once overt hepatic cirrhosis was established (Abramovitch S., Sharvit E. and others, 2015). Furthermore, vitamin D has been proposed as an effective modulator of insulin sensitivity in several experimental models and epidemiological data show the existence of a tight correlation between low circulating vitamin D levels and the presence of obesity, T2D and insulin resistance-related conditions (Bell N.H., and others, 1985).

2.1. MATERIAL AND METHOD

This chapter presents the research design which used in this study including the administrative arrangement, setting of the study, the sample of the study and criteria of the sample selection, the study instrument, pilot study, data collection, data analysis, and limitation of the study.

3.2. Liver function test

- Albumin, a protein made in the liver
- Total protein. This test measures the total amount of protein in the blood.
- ALP (alkalinephosphatase), ALT (alaninetransaminase), AST (aspartate aminotransferase), these are different enzymes made by the liver.
- Bilirubin, a waste product made by the liver.

ASPARTATE AMINOTRANSFERASE (AST) Test Procedure

A complete list of test parameters and operational procedure can be found in the User's Guide appropriate to the analyzer.

Materials Provided AST Reagent
-Stability of Final Reaction Mixture
The Beckman Coulter AU analyzer automatically computes every determination at the same time interval. Calibration of this AST procedure on the AU400/400e and AU600/640/640e is based upon the theoretical extinction coefficient for NADH, which has a molar absorptivity of 4960 at 340/380 nm. On the AU5800/2700/5400/680/480 it is based on experimental determination of the molar absorptivity at 340/660nm.

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. In addition, controls should be performed with each new lot of reagent, and after specific maintenance or troubleshooting steps described in the appropriate User's Guide. Quality control testing should be performed in accordance with regulatory requirements and each laboratory's standard procedure.

RESULTS

Automatically printed out for each sample in U/L at 37°C.

ALANINE AMINOTRANSFERASE (ALT) Test Procedure

A complete list of test parameters and operational procedure can be found in the User's Guide appropriate to the analyzer.

Materials Provided

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

Stability of Final Reaction Mixture

The Beckman Coulter AU analyzer automatically computes every determination at the same time interval. Calibration Calibration of this ALT procedure on the AU400/400e and AU600/640/640e is based upon the theoretical extinction coefficient of NADH, which has a molar absorptivity of 4960 at 340/380 nm. On the AU5800/2700/5400/680/480 it is based on experimental determination of the molar absorptivity at 340/660nm.

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. In addition, controls should be performed with each new lot of reagent, and after specific maintenance or troubleshooting steps described in the appropriate User's Guide. Quality control testing should be performed in accordance with regulatory requirements and each laboratory's standard procedure.

Results

Automatically printed out for each sample in U/L at 37°C.

TOTAL BILIRUBIN Test
PROCEDURE

A complete list of test parameters and operational procedure can be found in the User's Guide of the analyzer.

- Materials Provided Total Bilirubin Reagent
- Materials Required But Not Provided Chemistry Calibrator (Cat. No. DR0070)
- Stability of Final Reaction Mixture.

The Beckman Coulter AU analyzer automatically computes every determination at the same time interval.

- **Calibration**

The frequency of calibration is every 30 days. Calibration of this Total Bilirubin procedure is accomplished by use of the Chemistry Calibrator (Cat # DR0070), which is traceable to the National Institute of Standards and Technology (NIST) Standard Reference Material (SRM) 916a. Recalibration of this test is required when any of these conditions exist.

1. A reagent lot number has changed or there is an observed shift in control values.
2. Major preventative maintenance was performed on the analyzer.
3. A critical part was replaced.

- **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. In addition, controls should be performed after calibration, with each new lot of reagent, and after specific maintenance or troubleshooting steps described in the appropriate User's Guide. Quality control testing should be performed in accordance with regulatory requirements and each laboratory's standard procedure.

Results

Automatically printed out for each sample in mg/dL at 37°C. For SI units (µmol/L) the result must be multiplied by 17.1.

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

3.1 Diabetes tests (FBS , HbA1C)

A blood sugar test is simple procedure that allows the glucose to be measured in your blood. This test may be ordered by your doctor if it is suspected that your blood sugar level is abnormal. People with diabetes can greatly benefit from this test to monitor their blood sugar level.

3.1.1 Fasting Blood Sugar or FBS

The test is done in the morning, before the person has eaten. The range for normal blood glucose is 70 to 100 mg/dl. Readings between 100 and 126 mg/dl are considered as impaired fasting glucose or pre- diabetes. Diabetes is generally diagnosed when fasting blood glucose levels are 126 mg/dl or higher. this is a very simple blood test. Very simple complications may arise such as infection at the puncture site, bleeding, or difficulty in finding the vein.

Fasting test preparation

For a fasting blood glucose test, you can't eat or drink anything except water for 8 hours before your test. You may want to schedule a fasting glucose test first thing in the morning, so you don't have to fast during the day.

Fasting before a blood glucose test is important because it'll provide more accurate results that are easier for your doctor to interpret.

3.1.2 HbA1c

A hemoglobin A1c (HbA1c) test measures the amount of blood sugar (glucose) attached to hemoglobin. Hemoglobin is the part of your red blood cells that carries oxygen from your lungs to the rest of your body. An HbA1c test shows what the average amount of glucose attached to hemoglobin has been over the past three months. It's a three-month average because that's typically how long a red blood cell lives.

What happens during an HbA1c test?

A health care professional will take a blood sample from a vein in your arm, using a small needle. After the needle is inserted, a small amount of blood will be collected into a test tube or vial. You may feel a little sting when the needle goes in or out. This usually takes less than five minutes.

You don't need any special preparations for an HbA1c test. HbA1c results are given in percentages. Typical results are below.

- Normal: HbA1c below 5.7%

- Prediabetes: HbA1c between 5.7% and 6.4%
- Diabetes: HbA1c of 6.5% or higher.

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

- **High – Density Lipoprotein (HDL):** is a (good cholesterol) as help in removing chol. From tissue and in removing excess chol. From deposit the arteries so protect against heart diseases. High density lipoprotein (HDL) cholesterol Low serum concentrations of HDL-cholesterol are associated with increased risk for CHD. Coronary risk increases markedly as the HDL concentration decreases from 40- to 30 mg/dl. A low HDL-cholesterol concentration is considered to be a value below 35 mg/dl, and high HDL, >60 mg/dl. HDL-cholesterol values are also used in the calculation of LDL cholesterol.

- **Low – Density Lipoprotein (LDL):** is a (bad cholesterol) as it act opposite to the function of HDL so it will lead to chol. Deposit in the arteries and increased risk for heart diseases Most of the circulating cholesterol is found in three major lipoprotein fractions: very low density lipoproteins (VLDL), LDL and HDL.

[Total chol] = [VLDL-chol] + [LDL-chol] + [HDL-chol]

LDL-cholesterol is calculated from measured values of total cholesterol, triglycerides and HDL- cholesterol according to the relationship.

[LDL-chol] = [total chol] - [HDL-chol] - [TG]/5

LDL carries most of the circulating cholesterol in man and when elevated contributes to the development of coronary atherosclerosis. LDL-cholesterol is measured to assess risk for CHD and to follow the progress of patients being treated to lower LDL-cholesterol concentrations. Desirable levels of LDL-chol are those below 130 mg/dl in adults and 110 mg/dl in children.

Chylomicron: transport TG from Gut (Exogenous die try TG)

VLDL (15% chol.): transport TG from liver to tissue (Endogenous TG) LDL (60% chol.): transport chol. To tissue

HDL (25% chol.): transport chol. From tissue

PROCEDURE

1- Bring the Cholesterol MR Mono reagent and the cholesterol Standard (50 mg/dl) of the kit to room temperature

2- Pipette into labelled tubes

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

3- Mix and let the tubes stand for 10 minutes at room temperature Or 5 minutesat 37ºC.

4- Read the absorbance (A) of the supernatant and the standard At 500 nm against the reagent blank.

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.

RESULTS AND DISCUSSION

This chapter presents the findings of the data analysis systematically in tables and these correspond with the objectives of the study as follow.

4.1 Results

Table 4-1: Distribution of Study groups according to (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			
B MI	<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 Sig. at *P*>0.05; Testing based on a contingency coefficient test.

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	Ra n g e	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*						*Vi t. 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%).

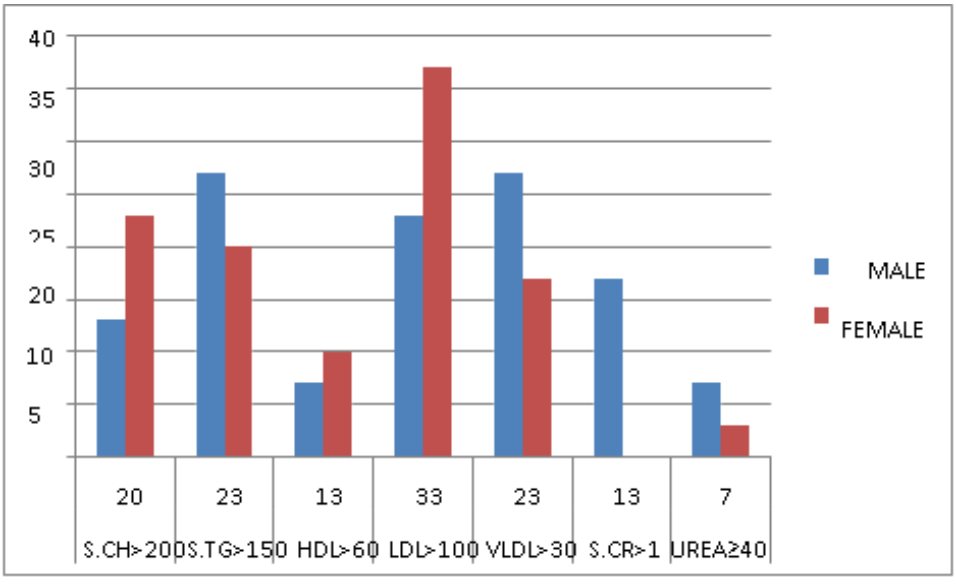

Figure 4-1: Correlation of BMI with HbA1c, FBS, TSB in all study groups.

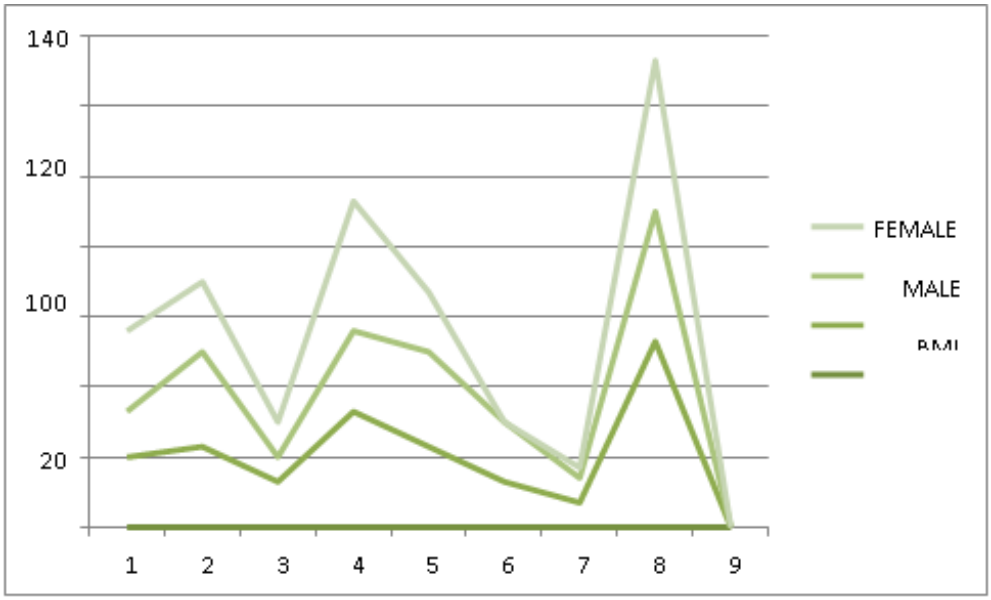

Figure 4-2: Correlation of gender & abnormal BMI with increase lipids and albumin in patients with.

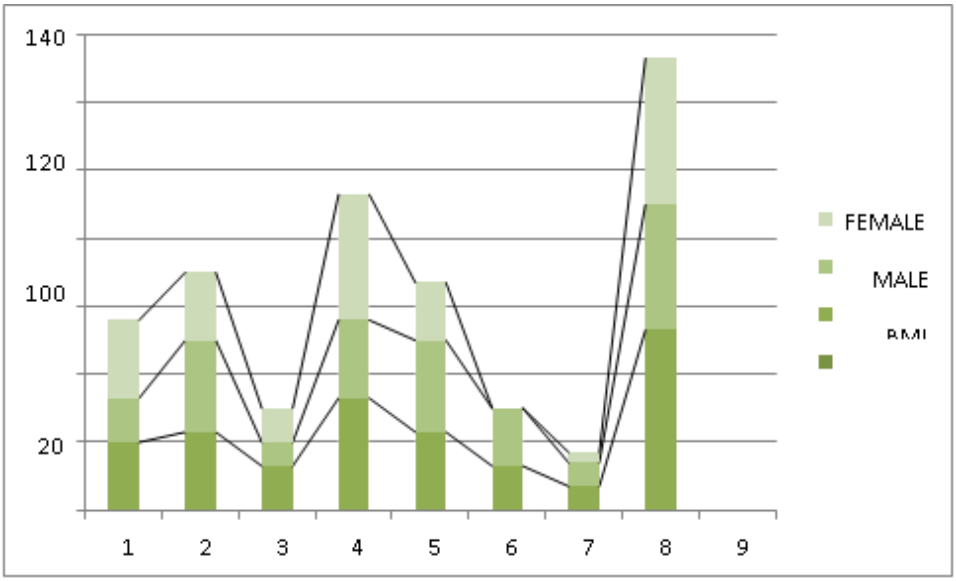

Figure 4-3: Correlation of gender & abnormal BMI with increase lipids and albumin in patients with.

3. RESULTS AND DISCUSSION

This chapter presents the findings of the data analysis systematically in tables and these correspond with the objectives of the study as follow: 4.1 Results Table 4-1: Distribution of study groups according to (gender, age, BMI).

5.1 CONCLUSION

1. NAFLD patients have low serum vitamin D concentrations, suggesting that vitamin D may have a role in the development of NAFLD. Future studies are recommended to determine the important therapeutic implications of vitamin D for the prophylaxis or the treatment 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 are more commonly have severe deficiency. Low serum levels of vitamin D have been observed in chronic liver diseases, especially with liver cirrhosis, while in patients with NAFLD, the data are still scanty.

3. The pleiotropic effects of vitamin D indicate a relationship between its deficiency and numerous chronic diseases, such as DM, cardiovascular, autoimmune and infectious diseases, several types of cancer and chronic liver diseases.

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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