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# Predictors of Fatty Liver Disease in Obese Ghanaian Adults with Periodontitis

Nafiu Amidu<sup>1\*</sup>, Serwaa Afia Opoku<sup>1</sup> and Peter Paul Mwinsanga Dapare<sup>1</sup>

<sup>1</sup>Department of Biomedical Laboratory Science, School of Allied Health Sciences, University for Development Studies, Tamale, Ghana.

## Authors' contributions

This work was carried out in collaboration among all authors. Authors NA and PPMD designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors NA, SAO and PPMD managed the analyses of the study. Authors NA and SAO managed the literature searches. All authors read and approved the final manuscript.

## Article Information

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Original Research Article

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# ABSTRACT

**Aim**: This study so to determine the factors associated with development of fatty liver disease in obese individuals with periodontitis.

Study Design: Hospital based cross-sectional study.

**Place and Duration of Study:** This hospital-based cross-sectional study was conducted at the Komfo Anokye Teaching Hospital (KATH), Ghana, from March, 2017 to February 2018.

**Methodology:** Eighty-seven (87) (29 males and 58 females) clinically diagnosed periodontal disease (PD) patients who were obese (BMI  $\geq$  30 kg/m<sup>2</sup>) were recruited. A self-designed semi-structured questionnaire was administered to each consented participant for socio-demographic characteristics. Oral hygiene data including dental visits, tooth extraction. Periodontal disease was diagnosed using Basic Periodontal Examination (BPE) by a qualified Restorative Dental surgeon. Blood samples were also collected for selected biochemical measurements.

**Results:** The prevalence of FLD in this study population is 37.9%. Higher age (RR = 8.4; 95% CI = 1.93-36.62; p = 0.005 for 31-40 years. and RR = 5.48; 95% CI = 1.41-21.30; p = 0.014 for > 40 years), being a female (RR = 2.55; 95% CI = 1.0-6.91; p = 0.035) and severity of periodontal

disease (RR = 5.95; 95% CI = 2.17-16.35; p = 0.001 for moderate periodontal state and RR = 7.00; 95% CI = 1.14-42.97; p = 0.036 for advanced periodontal state) significantly increased the risk of developing FLD among the study population. Also, hypertensives were 4 times more likely to develop FLD (RR = 4.24; 95% CI = 1.53-11.75; p = 0.006).

**Conclusion:** The risk factors for FLD among obese subjects with periodontal disease are age group (31 -40) years, being female, hypertension and severity of periodontal disease. This is important in the primary prevention and control of FLD among these subjects.

Keywords: Obesity; fatty liver disease; hypertension; periodontitis.

# 1. INTRODUCTION

Fatty liver disease (FLD) is on the rise and has become the most common form of liver disease worldwide [1]. The two major causes of FLD are alcohol consumption and overnutrition [2]. Excessive alcohol consumption can lead to alcoholic liver diseases (ALD) while overnutrition can induce non-alcoholic fatty liver disease (NAFLD) [1]. Non-alcoholic fatty liver disease (NAFLD) is characterized by a chronic and progressive hepatic pathology in which there is accumulation of fat in the liver without a history of drinking [3].

NAFLD is a heterogeneous disease and a considerable majority of individuals with NAFLD will not have any hepatic event or a shortened lifespan [4]. However, subjects with progressive NAFLD are at risk of increased hepatic, extrahepatic and overall morbidity and mortality [5]. Recently, studies have shown that the risk, clinical picture, and the disease burden of NAFLD is modified by genetic and epigenetic changes, lifestyle and environmental factors, inflammatory status, the well-being of the gut microbiota and hormonal balance [6,7].

In 1997, the WHO officially classified obesity as a chronic condition which fosters the development of other diseases, and is associated with increased mortality [8] without appropriate treatment. Obesity induces chronic systemic inflammation as a result of visceral adipose tissue [9]. Hypertrophic adipocytes weakly stimulate the local immune system leading to the release of inflammatory cytokines [10]. Depending on the severity of obesity, there is an increase in the number and size of adipocytes and a resultant increase in the amount of cvtokines produced and released in circulation which causes chronic inflammation [11]. This brings about hepatic inflammation and a resultant accumulation of fat, finally leading to NAFLD [12].

Periodontitis is a common public health problem worldwide [13], appearing as a highly prevalent, chronic inflammatory disease that affects everyday life negatively [14]. Periodontal disease arises when inflammatory reactions are induced by periodontal bacterial pathogens when the ecological balance of the oral cavity has been disrupted [15]. However, some studies have emphasized the association between FLD and periodontal disease [16] and also its link with obesity [17].

The severity and rate of progression of FLD may thus be compounded by the dual presence of periodontal disease and Obesity. In an individual however, this may be affected by some specific systemic risk factors, therefore understanding the factors that affect the severity and progression of FLD in obese individuals with periodontitis in our locality will provide important insight on primary prevention and management of the disease. This study therefore aimed at identifying the biochemical and clinical predictors of FLD in obese patients with periodontitis.

## 2. MATERIALS AND METHODS

## 2.1 Study Area and Design

This was a hospital-based cross-sectional study conducted from March 2017 to February 2018 at the Komfo Anokye Teaching Hospital (KATH) Kumasi, Ghana.

#### 2.2 Study Population

Eighty-seven (87) (i.e. 29 male and 58 female) obese subjects with clinically diagnosed periodontal disease (PD) were recruited for this study.

## 2.3 Data Collection

## 2.3.1 Questionnaire and anthropometry

A detailed self-designed semi-structured questionnaire was administered to each

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consented participant for socio-demographic characteristics such as age, gender, educational smoking, marital status, level. alcohol consumption and exercising. Oral hygiene data including dental visits, tooth extraction, tooth filling, tooth polishing, gum inflammation were also recorded. In this study, exercise was defined as any activity causing light perspiration or a slight to moderate increase in breathing or heart rate for at least 30 minutes. Alcohol intake was defined as the intake of at least one bottle of an alcoholic beverage per week. Participants were classified as smokers based on whether the respondent is in the habit of smoking at least one cigarette a day.

To determine the BMI, which was calculated by dividing weight (kg) by height squared (m<sup>2</sup>), participants were weighed to the nearest 0.1 kg on a bathroom scale (Zhongshan Camry Electronics Co. Ltd. Guangdong, China) barefooted while heights were measured using a wall-mounted ruler to the nearest 0.5 cm with participant standing upright and barefooted, with the heels put together and the head in the horizontal plane against the wall-mounted ruler.

Waist circumference was determined with a Gulick II spring loaded measuring tape (Gay Mills, WI). This was measured midway between the inferior angle of the ribs and the suprailiac crest. The hip circumference was measured at the outermost points of the greater trochanters.

Systolic and diastolic blood pressure measurement was taken using mercury sphygmomanometer and stethoscope according to the recommendation of the American Heart Association [18]. The measurement was repeated after 5 minutes rest interval and the average of the two values were recorded to the nearest 2.0 mmHg.

#### 2.3.2 Diagnosis and definitions

Periodontal disease was diagnosed using Basic Periodontal Examination (BPE) [19] by a qualified Restorative Dental surgeon. Radiograph features (i.e. pocket depth  $\geq$  3 mm, clinical attachment loss and radiographic bone loss) was used to stage the PD into Mild, Moderate and Advanced. Body mass index (BMI) was used to classify subjects as obese (i.e. BMI > 30 kg/m<sup>2</sup>). For the diagnosis of periodontitis, Patients were made to sit comfortably in a dental chair and rinsed their mouth with potable water in a disposable cup. They were then made to recline in an examination position and their mouths were dried with a gauze. A WHO 621 probe was then used to measure the pocket depth in six secants.

For each patient, the teeth were divided into six sides i.e., three buccal (mesio-buccal, buccal and disto-buccal) and three lingual (mesio-lingual, lingual and disto-lingual). WHO probe was placed into the gingival sulcus per patient and pockets depths as well as clinical attachment loss was recorded. Advanced Periodontitis was diagnosed when one or more teeth with Probing Pocket Depth (PPD) was  $\geq$  7 mm at any site, Moderate Periodontitis was diagnosed when one or more teeth with one or more teeth with Probing Pocket Depth (PPD) between 5 mm and 7 mm was recorded at any site while mild periodontitis was diagnosed when one or more teeth had a PPD between 3 mm and 5 mm.

## 2.3.3 Sampling and laboratory investigations

Fasting venous blood sample of 5 ml were drawn from each consented participant between 7 am and 10 am. The samples were dispensed into vacutainer® plain tubes and centrifuged within an hour at 500 g for 10 minutes. The serum was used for the biochemical assay.

Biochemical assays including lipid profiles, liver and cardiac enzymes on the serum were performed with the auto-analyser Roche COBAS Integra® 400 plus system according to the manufacturer's instructions (Roche Diagnostics, Germany, West Berlin).

## 2.3.4 Fatty liver index (FLI)

Fatty liver was assessed using the Bedogni, Bellentani [20] validated FLI whose accuracy in detecting fatty liver as compared to ultrasound was 0.84 (95% confidence interval [CI] 0.81– 0.87).

Fatty liver was estimated based on Body Mass Index (BMI), Waist Circumference (WC), serum Triglyceride (TG) concentration, serum Gama Glutamyl Transferase (GGT) concentration as follows:

 $FLI = \frac{e^{(0.953 \times In(TG) + 0.139 \times BMI + 0.718 \times In(GGT) + 0.053 \times WC - 15.74 \ \$}}{1 + e^{(0.953 \times In(TG) + 0.139 \times BMI + 0.718 \times In(GGT) + 0.053 \times WC - 15.74 \ \$}} X \ 100$ 

Based on the equation, the study participants were stratified into two groups: low risk of fatty liver (FLI < 60) and high risk of fatty liver (Fatty liver  $\geq$  60).

#### 2.4 Data Analysis

Data was entered into Microsoft Excel version 2013 and analysed using Systat for Windows, Version 13.0, (Systat Software, San Jose, CA). Data from the study was expressed as either mean $\pm$ SD (for continuous data) or proportions (for categorical data) and shown in tables. For continuous data, comparison between groups was done using unpaired t-test, whiles the Chi-square test was used for categorical variables. Logistic regression was used to assess the influence of different variables on Fatty liver disease. For all tests, a *P* < 0.05 was considered statistically significant at 95% confidence interval.

## 3. RESULTS

## 3.1 Socio-Demographic and Personal Hygiene Indicators among the Studied Participants

Of the 87 subjects included in the study, 29 (33.3%) were male and 58 (66.7%) were female. The study participants aged between of 19 to 60 vears and had an average age of 39.3±10.5 years. Most (62%) of the participants had a mild form of periodontal disease, 61% were married and all of them had attained at least basic education (Table 1). About 50% of the study participants indulged in alcoholic beverages, 60.9% were engaged in exercise and only 6.9% were smokers. On the personal and dental hygiene, more than half (59.8%) of the studied participants brushed their teeth once a day, about 45% visited the dental clinic based on the symptoms. 39.1% had had a tooth extraction before, 11.5% have had tooth filing before, 2.3% have had tooth polishing before and most (92%) of them have gum inflammation as shown in Table 1.

From this study population of obese subjects with periodontitis, the prevalence of FLD was 37.9% (i.e. 33 out of 87). When the studied participants were stratified based on the presence or absence of FLD, subjects with FLD were significantly older (P = 0.006) and had significantly higher proportion of participants with moderate (P = 0.001) and advanced (P = 0.02) periodontal disease. Significantly higher proportions of those with FLD were widowed (P =0.02) and had gum inflammation (P = 0.03) (Table 1). However, significantly higher proportion of those without FLD had mild periodontal disease (P = 0.0001), were single (P= 0.02) and smoked cigarette (P = 0.047) as shown in Table 1.

## 3.2 Anthropometric and Biochemical Parameters of the Studied Population Classified By FLD

As shown in Table 2, when the study population was stratified based on FLD, the mean SBP and DBP level was significantly higher (P = 0.006 and 0.001 respectively) in subjects with FLD (139.67 ± 19.00 mmHg and 90.73 ±10.33 mmHg respectively) compared to those without FLD (131.85 ± 17.37 mmHg and 83.31± 9.44 mmHg respectively). Also, all indicators of body weight including HC, AC, TC, waist-to-hip ratio, weight-to-hip ratio were significantly higher among those with FLD as compared to those without FLD as indicated in Table 2.

Variable	Total (n=87)	Without FLD (n=54)	With FLD (n=33)	<i>P</i> - value
Age	39.3 ± 10.5	36.9 ± 10.7	43.2 ± 8.9	0.006
Gender				
Female	58 (66.7)	32 (59.3)	26 (78.8)	0.06
Male	29 (33.3)	22(40.7)	7 (21.2)	
Periodontal State				
Mild	54 (62.1)	42 (77.8)	12 (36.4)	0.001
Moderate	27 (31.0)	10 (18.5)	17(51.5)	0.001
Advanced	6 (6.9)	1 (1.9)	5 (15.2)	0.02
Marital Status				
Single	26 (29.9)	21 (38.9)	5 (15.2)	0.02
Married	53 (60.9)	30 (55.6)	23 (69.7)	0.19
Divorced	5 (5.7)	4 (7.4)	1(3.0)	0.39
Widowed	3 (3.4)	0 (0)	3 (9.1)	0.02

Table 1. Socio-demographic and	personal hygiene of the studied	population stratified by FLD
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Variable	Total	Without FLD	With FLD	P- value
	(n=87)	(n=54)	(n=33)	
Educational				
Basic	19 (21.8)	11 (20.4)	8 (24.2)	0.79
Secondary	43 (49.4)	24 (44.4)	19 (57.6)	0.23
Tertiary	25 (28.7)	19 (35.2)	6 (18.2)	0.14
Frequency of Brushing	Per Day			
Once	52 (59.8)	33 (61.1)	19 (57.6)	0.74
Twice	35 (40.2)	21 (38.9)	14 (42.4)	
Alcohol Intake				
Yes	47 (54.0)	31 (57.4)	16 (48.5)	0.41
Smoking Habits				
Yes	6 (6.9)	6 (11.1)	0 (0)	0.047
Exercise				
Yes	53 (60.9)	32 (59.3)	21 (63.6)	0.68
Dental Visits				
Never	48 (55.2)	33 (61.1)	15 (45.5)	0.15
Symptom Based	39 (44.7)	21 (38.9)	18 (54.5)	
Tooth Extraction				
Yes	34 (39.1)	19 (35.2)	15 (45.5)	0.34
Tooth Filling				
Yes	10 (11.5)	5 (9.3)	5 (15.2)	0.40
Tooth Polishing				
Yes	2 (2.3)	1 (1.9)	1 (3.0)	0.72
Gum Inflammation				
Yes	80 (92.0)	47 (87.0)	33 (100)	0.03

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Categorical data are expressed as n (%) and continues data are expressed as mean ± SD

Variable	Total	Without FLD	With FLD	P- value
	(n=87)	(n=54)	(n=33)	
SBP	134.82 ± 18.30	131.85 ± 17.37	139.67 ± 19.00	0.006
DBP	86.13 ± 10.38	83.31 ± 9.44	90.73 ± 10.33	0.001
HC (cm)	105.05 ± 7.71	102.87 ± 6.19	108.61 ± 8.68	0.001
AC (cm)	32.31 ± 3.64	31.67 ± 4.05	33.35 ± 2.56	0.03
TC (cm)	58.31 ± 6.08	56.96 ± 6.18	60.50 ± 5.30	0.01
WHR	0.90 ± 0.77	0.88 ± 0.07	0.94 ± 0.07	0.001
Wt/H	0.76 ± 0.08	0.74 ± 0.08	0.78 ± 0.08	0.02
CK U/L	185.74 ± 108.73	177.69 ± 104.74	198.91 ± 115.39	0.38
LDH U/L	210.15 ± 50.20	204.00 ± 50.02	220.21 ± 49.60	0.14
T-Chol (mmol/L)	5.10 ± 1.10	5.01 ± 1.09	5.24 ± 1.11	0.34
HDL-c (mmol/L)	1.34 ± 0.31	1.36 ± 0.33	1.31 ± 0.26	0.42
LDL-c (mmol/L)	3.31 ± 0.87	3.23 ± 0.87	3.44 ± 0.88	0.28
VLDL-c (mmol/L)	2.03 ± 1.80	2.05 ± 1.79	2.00 ± 1.84	0.91
CR	3.98 ± 1.44	3.92 ± 1.68	4.09 ± 0.93	0.56
AST U/L	33.00 ± 7.54	22.52 ± 8.56	23.78 ± 5.51	0.45
ALT U/L	17.24 ± 10.34	17.17 ± 11.96	17.35 ± 7.10	0.94
ALP U/L	64.13 ± 21.36	64.11 ± 23.06	64.18 ± 18.59	0.98
FLI	50.00 ± 22.11	36.11 ± 14.39	72.72 ± 10.59	0.001

<b>Fable 2. Anthropometric and biochemical assa</b>	y of the studied population stratified B	y FLD
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 SBP-Systolic Blood Pressure, DBP- Diastolic Blood Pressure, HC- Hip Circumference, AC- Arm Circumference, TC – Thigh Circumference, WHR – Waist Hip Ratio, Wt/H- Weight to Hip Ratio, CK- Creatinine Kinase, LDH – Lactate Dehydrogenase, T-Chol – Total Cholesterol, HDL-c – High Density Lipoprotein cholesterol, LDL-c- Low Density Lipoprotein cholesterol, VLDL-c – Very Low Density Lipoprotein cholesterol, CR – Coronary Risk, AST – Aspartate Transaminase, ALT – Alanine Transaminase, ALP- Alanine Phosphatase, FLI – Fatty Liver Index

## 3.3 Determinants of FLD among the Obese Periodontal Subjects

The effect of different socio-demographic and personal hygiene variables on the FLD risk is shown in Table 3. Higher age (RR = 8.4; 95% CI = 1.93-36.62; P = 0.005 for 31-40 years. and RR = 5.48; 95% CI = 1.41-21.30; P = 0.01 for > 40 years), being a female (RR = 2.55; 95% CI = 1.0-6.91; P = 0.04) and severity of periodontal disease (RR = 5.95; 95% CI = 2.17-16.35; P = 0.001 for moderate and RR = 7.00; 95% CI = 1.14-42.97; P = 0.04 for advanced) and

Hypertension (RR=4.24; 1.53-11.75; *P*=0.006) significantly increased the risk of developing FLD among obese subjects with periodontal disease from the univariate analysis in Tables 3 and 4.

After adjusting for confounding variables, which includes age, gender, periodontal state and hypertension, the risk factors for FLD among obese subjects with periodontal disease are age (31- 40 years), gender (females more at risk), hypertension and severity of periodontitis (Tables 3 and 4).

Table 2. Socio-demographic risk factors for FLD among th	e stuaiec	i population
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Variable	RR (95% CI)	P-value	aRR (95% CI)	<i>P</i> -value
Age (Years)				
≤ 30				
31-40	8.40 (1.93-36.62)	0.005	4.76 (1.01-22.81)	0.04
> 40	5.48 (1.41-21.30)	0.01	2.77 (0.63-12.14)	0.18
Gender				
Female	2.55 (1.00-6.91)	0.03	4.00 (1.19-13.41)	0.03
Male				
Periodontal State				
Mild				
Moderate	5.95 (2.17-16.35)	0.001	3.71 (1.25-11.01)	0.01
Advance	7.00 (1.14-42.97)	0.03	5.59 (1.01-38.50)	0.04
Marital Status				
Single				
Married	1.84 (0.74-4.60)	0.19	1.04 (0.33-3.22)	0.95
Frequency of Brushi	ng Per Day			
Once	0.86 (0.36-2.08)	0.74	0.98 (0.34-2.79)	0.97
Twice				
Highest Education				
Basic	2.18(0.60-7.98)	0.24	2.32 (0.50-10.80)	0.26
Secondary	2.28 (0.76-6.85)	0.14	2.45 (0.66-9.16)	0.18
Tertiary				
Alcohol Intake				
Yes	0.70 (0.29-1.67)	0.42	0.50 (0.17-1.48)	0.21
Exercise				
Yes	0.83 (0.34-2.03)	0.68	1.43 (0.45-4.57)	0.55
Dental Visits				
Symptom Based	1.89 (0.79-4.53)	0.15	0.62 (0.19-2.08)	0.44
Never				
Tooth Extraction				
Yes	1.54 (0.63-3.72)	0.34	0.53 (0.16-1.77)	0.29
Tooth Filling				
Yes	1.75 (0.47-6.58)	0.41	0.59 (0.12-2.83)	0.51
Tooth Polishing				
Yes	1.66 (0.10-27.41)	0.72	1.91 (0.09-40.10)	0.67

Variable	RR (95% CI)	<i>P</i> -value	aRR (95% CI)	<i>P</i> -value
Hypertension				
Yes	4.24 (1.53-11.75)	0.006	2.46 (1.17-7.83)	0.03
CK (U/L)				
> 150	1.33 (0.56-3.17)	0.52	0.62 (0.20-1.93)	0.4
≤ 150				
LDH (U/L)				
> 200	2.19 (0.90-5.32)	0.09	1.73 (0.57-5.24)	0.33
≤ 200				
T-Chol (mmol/L)				
≥ 6.21	1.43 (0.40-5.11)	0.58	1.40 (0.32-6.14)	0.65
< 6.21				
HDL-c (mmol/L)				
< 1.03	1.49 (0.46-4.90)	0.51	1.05 (0.25-4.42)	0.95
≥ 1.03				
LDL-c (mmol/L)				
> 4.14	1.03 (0.31-3.45)	0.97	0.87 (0.20-3.68)	0.85
≤ 4.14				
AST (U/L)				
> 30	1.75 (0.47-6.58)	0.41	2.45 (0.46-12.98)	0.29
≤ 30				

Table 3. Biochemical and clinical determinants of FLD among the studied population

CK- Creatinine Kinase, LDH – Lactate Dehydrogenase, T-Chol – Total Cholesterol, HDL-c – High Density Lipoprotein cholesterol, LDL-c- Low Density Lipoprotein cholesterol, VLDL-c – Very Low-Density Lipoprotein cholesterol, CR – Coronary Risk, AST – Aspartate Transaminase, ALT – Alanine Transaminase, ALP- Alanine Phosphatase

## 4. DISCUSSION

Periodontitis is a common public health problem worldwide [13], which arises when inflammatory reactions are induced by periodontal bacterial pathogens. Obesity has also been implicated in the pathogenesis of FLD since it induces chronic systemic inflammation as a result of visceral adipose tissue [9]. Some studies have emphasized the association between FLD, obesity and periodontal disease [16,17] and a dual occurrence of both obesity and periodontitis may thus compound the FLD situation. These may however be affected by some systemic risk factors specific to individuals and therefore understanding the determinants of FLD among obese individuals with periodontitis in our locality will provide important insight on primary prevention and management of the disease. This study therefore aimed at identifying the biochemical and clinical predictors of FLD in obese patients with periodontitis.

The prevalence of FLD among obese individuals with periodontitis was 37.9%. This was higher than the speculated global prevalence of FLD, which is 20-30% [21]. This study showed an increased prevalence in fatty liver disease as the lowest prevalence of FLD has been reported to

be around (13.48%) in Africa, 31.79% in the Middle East and 30.45% in South America [22] but lower than the reported global prevalence (57-74%) [3]. Interestingly, a prevalence of 4.5% has been reported by [23] among 44 Nigerians in an urban hospital based study. The observed differences could be due in part to the differences in study population, diagnostic modality and racial and or ethnicity disparity.

In this study age was seen to influence the occurrence of FLD in obese individuals with periodontitis. It was observed that, obese individuals with periodontitis between the ages of 31- 40 years are more at risk of developing FLD. Similar findings were reported by Fan, Zhu [24]. However some contrasting findings reported peak prevalence in the fourth decade onwards i.e. 40-60 years [25]. FLD risk factors may vary across different ages due to variation in endocrine function and fat distribution, this could account for the difference in these findings [26]. Furthermore, it has been reported that in low and middle income countries like Ghana, the distribution of adult nutritional imbalance is shifting from undernutrition to overnutrition leading to obesity and increasing the risk of FLD thereof [27].

This study also reports that, in obese individuals with periodontitis, FLD was more associated with female. This finding conforms with data from Western and Asian populations [28]. However, other studies report that FLD is more common in men [29]. The reasons for the reported gender related difference in the association with FLD in obese individuals is not clear and needs to be fully studied. However, physiological pathways such as the differences in body fat distribution and the influence of gonadal hormones have sometimes been used to explain gender differences in the pathogeneses of some diseases.

Hypertension was also observed as a risk factor for developing FLD in obese subjects with periodontitis from this study. This is similar to findings of Fan, Saibara [30]. This may be seen as an effect rather than a cause, due in part to the increased risk of developing hypertension as a result of obesity [31]. Obesity causes accumulation of lipids in the blood vessels and subsequent hardening, resulting in narrowing of blood vessels and development of atherosclerosis [32]. Individuals with FLD do not not only accelerate the development of severe liver diseases but also the increase of blood pressure and vice versa among different kinds of population in various regions [33].

strong severity-dependent association The between periodontitis state and FLD from this study learns credence to the fact that a history of periodontitis is essential in predicting FLD. The principal mechanism is linked to high level of inflammation which increases the chance of initiation and progression of periodontitis and consequently to an amplified inflammatory response [34]. Besides, enhanced permeability of the epithelial lining of the gut can change its microbial composition [35], which can reach the liver via portal vein. This can result in the activation of the resident cells bv proinflammatory factors that would eventually activate cytokines and, reactive oxygen species (ROS) that set the stage for liver injury [36].

## **5. CONCLUSION**

This study highlights a worrying prevalence of FLD among obese subjects with periodontitis. It also suggests that the risk factors associated with FLD among obese subjects with periodontitis include age, gender, hypertension and the severity of the periodontitis. The methodology of this study is however limited in

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showing a causal relationship between these factors and the occurrence of FLD in the subjects of this study and the fatty liver index as used in this study though highly correlative with sonographically confirmed FLD, is not always definite evidence of FLD.

## CONSENT

All authors declare that verbal informed consent was obtained from the patients before being included in the study. Participation was voluntary and non-consenting participants were assured that their non participation in the study will not affect the care given them at the facility.

## ETHICAL APPROVAL

The study was approved by the Committee on Human Research Publications and Ethics of Komfo Anokye Teaching Hospital and School of Medical Sciences, Kwame Nkrumah University of Science and Technology.

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#### **COMPETING INTERESTS**

Authors have declared that no competing interests exist.

## REFERENCES

- Gao B, Bataller R. Alcoholic liver disease: Pathogenesis and new therapeutic targets. Gastroenterology. 2011;141(5):1572-1585.
- Mills, et al. Comparison of the natural history of alcoholic and nonalcoholic fatty liver disease. Current Gastroenterology Reports. 2005;7(1):32-36.
- Angulo P. Nonalcoholic fatty liver disease. New England Journal of Medicine. 2002;346(16):1221-1231.
- Rinella M, Charlton M. The globalization of nonalcoholic fatty liver disease: Prevalence and impact on world health. Hepatology. 2016;64(1):19-22.
- Ekstedt M, et al. Fibrosis stage is the strongest predictor for disease-specific mortality in NAFLD after up to 33 years of follow-up. Hepatology. 2015;61(5):1547-1554.

- Buzzetti E, Pinzani M, Tsochatzis EA. The multiple-hit pathogenesis of non-alcoholic fatty liver disease (NAFLD). Metabolism. 2016;65(8):1038-1048.
- Petäjä E, Yki-Järvinen H. Definitions of normal liver fat and the association of insulin sensitivity with acquired and genetic NAFLD—a systematic review. International Journal of Molecular Sciences. 2016;17(5): 633.
- World Health Organization. Obesity and overweight; 2015.
- 9. Festi D, et al. Hepatic steatosis in obese patients: Clinical aspects and prognostic significance. Obesity Reviews. 2004;5(1): 27-42.
- Lynch L, et al. Adipose tissue invariant NKT cells protect against diet-induced obesity and metabolic disorder through regulatory cytokine production. Immunity. 2012;37(3):574-587.
- 11. Wensveen FM, et al. Interactions between adipose tissue and the immune system in health and malnutrition. In Seminars in Immunology. Elsevier; 2015.
- Bugianesi E, McCullough AJ, Marchesini G. Insulin resistance: a metabolic pathway to chronic liver disease. Hepatology. 2005;42(5):987-1000.
- Batchelor P. Is periodontal disease a public health problem? British Dental Journal. 2014;217(8):405.
- O'Dowd LK, et al. Patients' experiences of the impact of periodontal disease. Journal of Clinical Periodontology. 2010;37(4):334-339.
- 15. Gheorghe DN, et al. Hepatitis C infection and periodontal disease: Is there a common immunological link? Journal of Immunology Research; 2018.
- Han Sun, Yang. Interaction between periodontitis and liver diseases. Biomed Rep. 2016;5(3):267-76.
- 17. Genco, et al. A proposed model linking inflammation to obesity, diabetes and periodontal infections. Journal of Periodontology. 2005;76:2075-2084.
- Kirkendall WM, et al. Recommendations for human blood pressure determination by sphygmomanometers. Circulation. 1967; 36(6):980-988.
- 19. British Society of Periodontology, Basic Periodontal Examination (BPE). British Society of Periodontology; 2011.
- 20. Bedogni G, et al. The fatty liver index: A simple and accurate predictor of hepatic

steatosis in the general population. BMC Gastroenterology. 2006;6(1):33.

- Williams CD, et al. Prevalence of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis among a largely middle-aged population utilizing ultrasound and liver biopsy: A prospective study. Gastroenterology. 2011;140(1):124-131.
- 22. Younossi ZM, et al. Global epidemiology of nonalcoholic fatty liver diseasemeta-analytic assessment of prevalence, incidence and outcomes. Hepatology. 2016;64(1):73-84.
- Asabamaka Onyekwere C, Ogbera AO, Balogun BO. Non-alcoholic fatty liver disease and the metabolic syndrome in an urban hospital serving an African community. Annals of Hepatology. 2016;10(2):119-124.
- 24. Fan JG, et al. Prevalence of and risk factors for fatty liver in a general population of Shanghai, China. Journal of Hepatology. 2005;43(3):508-514.
- 25. Ayonrinde OT, et al. Gender-specific differences in adipose distribution and adipocytokines influence adolescent nonalcoholic fatty liver disease. Hepatology. 2011;53(3):800-809.
- 26. Takasugi M, et al. Age-and sex-dependent DNA hypomethylation controlled by growth hormone in mouse liver. Mechanisms of Ageing and Development. 2013;134(7-8): 331-337.
- Ofori-Asenso R, et al. Overweight and obesity epidemic in Ghana—a systematic review and meta-analysis. BMC Public Health. 2016;16(1):1239.
- Wang Z, et al. Prevalence and associated metabolic factors of fatty liver disease in the elderly. Experimental Gerontology. 2013;48(8):705-709.
- Xu C, et al. Prevalence and risk factors for the development of nonalcoholic fatty liver disease in a nonobese Chinese population: the Zhejiang Zhenhai study. American Journal of Gastroenterology. 2013;108(8): 1299-1304.
- Fan JG, et al. What are the risk factors and settings for non-alcoholic fatty liver disease in Asia–Pacific? Journal of Gastroenterology and Hepatology. 2007;22(6): 794-800.
- 31. Jiang SZ, et al. Obesity and hypertension. Experimental and Therapeutic Medicine. 2016;12(4):2395-2399.

- 32. Defronzo RA. Is insulin resistance atherogenic? Possible mechanisms. Atheroscler Suppl. 2006;7(4):11-5.
- Ryoo JH, et al. Clinical association between non-alcoholic fatty liver disease and the development of hypertension. Journal of Gastroenterology and Hepatology. 2014;29(11):1926-1931.
- 34. Shaddox L, et al. Hyper-responsive phenotype in localized aggressive

periodontitis. Journal of Dental Research. 2010;89(2):143-148.

- 35. Arimatsu K, et al. Oral pathobiont induces systemic inflammation and metabolic changes associated with alteration of gut microbiota. Scientific Reports. 2014;4: 4828.
- Imajo K, et al. Microbiota and nonalcoholic steatohepatitis. In Seminars in Immunopathology. Springer; 2014.

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