

Rocz Panstw Zakl Hig 2022;73(3):325-332

https://doi.org/10.32394/rpzh.2022.0223

MODELS TO PREDICT NON-ALCOHOLIC FATTY LIVER DISEASE LINKED TO OBESITY IN MOROCCO

http://wydawnictwa.pzh.gov.pl/roczniki_pzh/

Habiba Liba^{1,}^(b), Rekia Belahsen^{1,}^(b)

¹Laboratory of Biotechnology, Biochemistry & Nutrition, Training and Research Unit on Nutrition & Food Sciences, Chouaib Doukkali University, Faculty of Sciences, El Jadida, Morocco

ABSTRACT

Background. The prevalence, risk factors and screening for the problem of non-alcoholic fatty liver disease linked to obesity are not well known in Morocco. The diagnosis of this disease by biopsy is invasive and the assessment of its severity by ultrasound shows variability in observation.

Objective. The aim of this retrospective study is to estimate the prevalence of NAFLD linked to obesity, to determine the risk factors associated with it and to develop a non-invasive procedure as a method of diagnosing this disease in Morocco. **Material and Methods.** It's a retrospective study. The collection of anthropometric, clinical, biochemical, and radiological data over a period from 2014 to 2018 were captured from registers of patients at the Med VI University Hospital in Marrakech. Data were analyzed using SPSS version 26 software. Descriptive statistics were presented using frequencies and means +/- standard deviation to describe categorical and numeric data respectively. Pearson's chi-square test was used to test the association between categories of two independent samples. Multinomial logistic regression is used to find disease risk factors and models to predict non-alcoholic fatty liver disease (NAFLD) linked to obesity in Morocco. **Results.** Gender, increased age, body mass index, alanine aminotransferase, triglycerides, C-reactive protein, alkaline

Results. Gender, increased age, body mass index, alanine aminotransferase, triglycerides, C-reactive protein, alkaline phosphatase, gamma-glutamyl transferase were significantly correlated with NAFLD and its evolvement.

Conclusion. The prevalence of NAFLD linked to obesity is an alarming problem in Morocco. It was 83.5%. Age, gender, body mass index, alanine aminotransferase, triglycerides, C-reactive protein, alkaline phosphatase and gamma-glutamyl transferase are risk factors for NAFLD and its severity. It were used to develop two algorithms that can be used, as a more objective and non-invasive screening method for NAFLD.

Key words: NAFLD, obesity, prevalence, non-invasive screening method

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is one of the most common liver diseases in the world. It is characterized by the accumulation of triglycerides in the liver without significant alcohol consumption (<20 g/day for women and <30 g/day for men) [1]. The disease has been associated with several risk factors including metabolic syndrome, overweight or obesity, diabetes mellitus, insulin resistance, drug use and environmental factors [2]. It is a syndrome that encompasses several liver pathologies ranging from simple steatosis, when fat accumulates in the liver, to non-alcoholic steatohepatitis (NASH), when the fat accumulated in the liver causes inflammation of the latter, to fibrosis and cirrhosis, when chronic inflammation progresses to advanced scarring of the liver [3]. NAFLD is most often clinically silent, but may be manifested by the presence of symptoms such

as asthenia or a feeling of discomfort in the right upper quadrant [4]. Liver enzymes are found to be normal in over 75% of cases. However, an increase in alanine aminotransferase (ALAT), triglycerides (TG) and gamma-GT (GGT) and an ASAT/ALAT ratio of less than 1 is found in about a quarter of cases. This ratio tends to become greater than 1 with the progression of the disease and the development of cirrhosis [4]

So far, liver biopsy has been the ultimate standard for the detection of fatty liver disease and the differentiation between its stages. However, the relatively high costs, patient discomfort, sampling variability, inter- and intra-observer variability and risk of complications as well as invasiveness make it unsuitable for screening for NAFLD [4]. Thus, despite the variability in the interpretation of images even in the same person, ultrasound is the first-line imaging examination used in clinical practice, for the diagnosis of NAFLD due to its wide availability and its lower

Corresponding author: Rekia Belahsen, Training and Research Unit on Nutrition & Food Sciences, Chouaib Doukkali University, Faculty of Sciences, El Jadida, 24000 Morocco, e-mail: b.rekia@gmail.com or rbelahsen@yahoo.com © Copyright by the National Institute of Public Health NIH - National Research Institute cost. The presence and grade of NAFLD was defined by ultrasound on a scale of 0 to 3 (0 = no abnormality, 1 = mild, 2 = moderate, 3 = severe) [5]. However, NAFLD can be detected by computed tomography without contrast, but it is less used as a screening test due to its lower sensitivity and the patient's exposure to radiation [6], whereas transient elastography (TE) is used for assessment of fibrosis [7]. On the other hand, magnetic resonance imaging (MRI) has a better sensitivity for the assessment of NAFLD but this modality cannot differentiate NAFLD from NASH. In addition, MRI combined with elastography (MRE) is a better method of identifying degrees of fibrosis in patients with NAFLD. However, MRI with or without MRE is expensive [8].

Due to the lack of a simple and non-invasive diagnostic test, the prevalence of NAFLD in the general population is uncertain and difficult to assess accurately [9].

Epidemiological data show that the global prevalence of NAFLD in different populations is estimated at 30% in the United States, 32% in the Middle East, 30% in South America, 27% in Asia, 24% in Europe and 13% in Africa [2]. This disease is associated with obesity and metabolic disturbances such as insulin resistance (IR), type 2 diabetes mellitus (T2DM) and dyslipidemia [9, 10]. NAFLD is itself an independent risk factor for cardiovascular disease, leading to increased all-cause mortality and increased liver-related mortality [10]. In the obese population, the prevalence of NAFLD is estimated at around 60 to 95% and that of NASH at 18.5% in obese patients against 3% in non-obese [11].

Furthermore, the global prevalence of obesity has been estimated at 51.34% and 81.83% in patients with NAFLD and NASH, respectively [2]. In this study, overweight and general obesity were defined by BMI and abdominal obesity, representing the percentage of abdominal fat mass, was measured by waist circumference according to the World Health Organization (WHO) [12]. In the NAFLD patients in this study, the determination of the prevalence of comorbidities showed that the metabolic syndrome (MetS) was prevalent in 42% of the NAFLD subjects; 42% had hyperlipidemia; 51% were obese; 39% were hypertensive and 22% had diabetes [2, 9].

In Morocco, while there is no data on the prevalence of NAFLD in Morocco, the country has 53% of its adult population over 18 years of age with overweight, 20% obesity, 10.5% with high blood cholesterol, 29.3% hypertension, 10.6% a diabetes and 10.4% are prediabetic [13,14]. In addition, ad hoc surveys carried out in different regions of the country have also reported an increasing trend in the prevalence of all these risk factors as well as that of the metabolic syndrome [14, 15]. With a view to improving the quality of care for people with non-alcoholic fatty liver disease linked to obesity, the objective of this retrospective study is to estimate the prevalence of NAFLD linked to obesity, to determine the risk factors associated with it and to develop a non-invasive procedure as a method of diagnosing this disease in Morocco.

MATERIAL AND METHODS

After informed consent, the collection of anthropometric, clinical, biochemical, and radiological data over a period from 2014 to 2018 were captured from registers of patients at the Med VI University Hospital in Marrakech. Incomplete records, records of patients with tuberculosis or seropositive hepatitis B or C, those with a history of another type of hepatitis or *Wilson's* disease, and those with a history of alcohol consumption were excluded.

The variables selected are age, sex, body mass index, waist circumference, having high blood pressure, diabetes and dyslipidemia. Parameters concerning laboratory analyzes, in particular serum levels of alkaline phosphatase (ALP), gamma-glutamyl transpeptidase (GGT), alanine aminotransferase (ALAT)), aspartate aminotransferase (ASAT), fasting glucose (FG), total cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL) and C-reactive protein (CRP) were also collected. However, measurement of insulin resistance was not cited due to lack of data in the study sample. The weight status of the study population is assessed by calculating the body mass index (BMI) by dividing the weight in kilograms by the square of the height in meters (kg/m^2) . Overweight was defined by a BMI greater than or equal to 25 and less than 30 and obesity by a BMI greater than or equal to 30 [12]. Abdominal obesity, reflecting abdominal fat mass, is measured by waist circumference (WC) in cm. Men with a waist circumference <94 were classified as normal weight and WC 94-101.9 overweight and those with WC \geq 102 cm obese. Women were classified into the same obesity categories based on WC <80, 80- $87.9 \text{ and } \ge 88 \text{ cm} [12].$

Blood pressure was measured after rest with a mercury sphygmomanometer. Based on WHO guidelines, a threshold of 140/90 mmHg for hypertension has been used [16]. Participants whose blood pressure was 140/90 mmHg or less were considered normotensive, while those with higher values or who reported taking antihypertensive drugs were classified as hypertensive [17]. Diabetes mellitus is characterized by hyperglycemia. Glycated hemoglobin is a simple marker of blood sugar and its value is usually expressed as a percentage. A level greater than or equal to 6.5% determined by high performance liquid chromatography (HPLC), twice, has been included in the diagnostic criteria for diabetes by the American Diabetes Association [18]. Normal blood glucose values are less than 100 mg/ dl in the fasting state and less than 140 mg/dl at the second hour of oral hyperglycemia according to WHO recommendations [18].

Laboratory analyzes of blood taken by venous blood puncture, mainly after overnight fast, provided information on the various functions of the liver. The increase in these indicators is a sign of an anomaly. The ALAT and ASAT transaminases remain renowned for their sensitivity; the specificity for the liver becomes excellent beyond their increase by a factor of 10, ie 300 to 400 U L. For usual values <35 (men) and <32 (women) U / L for ALAT; <43 (male) and <32 (female) U / L for GGT; <115 U / L (male and female) for serum ALP; 4.25-6.55 (man) and 4.05-5.55 (woman) mmol/l for cholesterol (serum) [19].

Statistical analyses

Data entry and analysis was performed using SPSS version 26 software. Descriptive statistics were presented using frequencies and means +/- standard deviation to describe categorical and numeric data respectively. Pearson's chi-square test was used to test the association between categories of two independent samples. Multinomial logistic regression is used to find disease risk factors that characterize a group of obese subjects with grade 1 NAFLD and those related to grade 2 and 3 NAFLD. Odds ratio (OR) and their confidence intervals (CI) were used to assess the risk of NAFLD if a certain factor is present. The significance level used is 0.05 (p-value).

RESULTS

The demographic and laboratory characteristics of the study sample are summarized in Table I. The results show that approximately 50% of the study participants presented NAFLD, among them 42.5% were with steatosis, 3.5 with fibrosis and 4.4 have cirrhosis. According to gender, women were more affected than men by steatosis (82.3% vs. 17.7%) and cirrhosis (70.6% vs. 29.4%). As for fibrosis, it was more present in men (64.3%) than in women (35.7%). In general, the average age of the participants was 45 \pm 15.03. The subjects without NAFLD have a mean age of 40.68 \pm 14.56 years, those with NAFLD grade 1 were 48.65 \pm 14.53 years old, and those with grade 2-3 have mean age of 43.00 \pm 11.93 and 59.94 \pm 10.19 respectively.

Among people with steatosis, 51.9% were over 50 years old, 36.8% between 18 and 50 years old and 20% were under 18 years old. A statistically significant association is found between age and the disease. *Pearson's Chi*-square test is indeed 34.55 and

the likelihood ratio is 36.64 with a value of p = 0.000 < 0.05.

The body mass index (BMI) means for the steatosis grades 0, 1, 2 and 3 were 40.12 ± 7.83 ; 39.78 ± 11.09 ; 32.97±8.23 and 28.14±6.36 respectively. The prevalence of simple NAFLD increased from 6.1% in people with a normal BMI to 10.4% in overweight people and to 83.5% in obese people. Fibrosis was prevalent in 14.3% of the subjects with normal weight or overweight while this prevalence achieved 71.4% in obese people. Concerning the cirrhosis, it was present in 35.3% of the normal weight, 29% of the overweight and in 35.3% of the obese people. The results also show that the BMI decreases for fibrosis and cirrhosis (Table 2). The value of the Pearson's Chi-square test shows a statistically significant association between BMI and NAFLD (of 52,162; p=0.000 <0.05) with a *Chi*-squared likelihood test value of 37,286 (p = 0.00 < 0.05).

The average waist circumference according to steatosis grades 0, 1 and 2-3 was respectively 115.63 ± 17.41 , 113.03 ± 19.48 and 90.64 ± 13.20 in women and 116.87 ± 19.31 , 124.83 ± 32.10 , 95.28 ± 18.13 respectively in men . A significant association between waist circumference and NAFLD was found (*Pearson's Chi*-square tests were 38.7 and 18.18 with p<0.05) (Table 1).

The distribution of the sample investigated according to the presence of chronic diseases shows a coexistence of NAFLD and type 2 diabetes in 47.2% of the subjects. However, the association of both diseases is not statistically significant (p = 0.327) according to the value of *Pearson's Chi*-square test (2.236). Furthermore, the dyslipidemia present in 13.3% of NAFLD patients was significantly associated with NAFLD (p-value = 0.002; Pearson's Chisquare value = 12.084). On the other hand, although not significantly associated with NAFLD, anemia was present in 8.2% of NAFLD patients (Pearson's Chi-square test = 3.295; p = 0.193 > 0.05). Added to these chronic diseases, more than half of people with NAFLD (52%) had high blood pressure, the association between these two diseases (NAFLD and hypertension) was however not statistically significant (chi-square test of *Pearson* = 3.579; p = 0.167 > 0.05%).

Risk factors for NAFLD

Multinomial logistic regression was used to determine the risk factors for NAFLD characterizing a group of obese subjects with grade 1 NAFLD and those related to NAFLD with grade 2 and 3, from anthropometric and biochemical data on a sample of 386 Moroccans. Thus, increased age, C-reactive protein and triglycerides (TG) had significant effects on the risk of NAFLD regardless of the degree of the disease. Male gender, body mass index, ALAT, ALP,

	Gra	ades of NAFLD	N (%)		
	Grade 0 191(49.5)	Grade 1 164(42.5)	Grade 2 14(3.5)	Grade 3 17(4.4)	Total 386
Age	40.68±14.56	48.65±14.53	43.00 ± 11.93	59.94±10.19	45±15.03
Age categories		1	1	1	1
<18yrs	12(80)	3(20)	0(0.0)	0(0)	15(3.9)
[18-50]	121(57.9)	77(36.8)	9(4.3)	2(1)	209(54.00)
≥50	58(35.8)	84(51.9)	5(3.1)	15(9.3)	162(42.10)
Gender					320
Female	168(88)	135(82.3)	5(35.7)	12(70. 6)	66
Male	23(12)	29(17.7)	9(64.3)	5(29. 4)	
BMI (kg/m2)	40. 12 ± 7.83	$39.78 \pm 11,09$	32.97 ± 8.23	28.14±6.36	
BMI categories		1	1	1	1
Normal	4(2.1)	10(6.1)	2(14.3)	6(35.3)	22(5.7)
Overweight	9(4.7)	17(10.4)	2(14.3)	5(29.4)	33(8.5)
Obesity	178(93.2)	137(83.5)	10(71.4)	6(35.3)	331(85.8)
WC (cm)					
Males	116.87±19.31	124.83±32.10	95.56±18.32	95±17.93	
Females	115.63±17.41	113.03±19.48	91.20±19.10	90.08±7.30	
WC categories					
	2(14.3)	6(42.9)	2(14.3)	4(28.6)	14(21 21)
94-102	3(25)	5(417)	4(33.3)	0(0)	12(18.18)
>102	18(45)	18(45)	3(7.5)	1(2,5)	40(60.61)
Total	23(34.8)	29(43.9)	9(13.6)	5(7.6)	66(100)
Females	20(0.110)		,(1010)	0(10)	00(100)
<80	3(25%)	7(58.3%)	1(8.3%)	1(8.3%)	12(3.75%)
80-88	1(5.6%)	12(66.7%)	2(11.1%)	3(16.7%)	18(5.6 2%)
≥88	164(56.6%)	116(40%)	2(0.7%)	8(2.8%)	290(90.63%)
Total	168(52.5%)	135(42.2%)	5(1.6%)	12(3.8%)	320(100%)
ASAT (unit/l)	22.05±12.14	31.43±23.37	58.37±33.75	45.22±36.35	
ALAT (unit/l)	22.15±14.10	29.48±21.15	96.89±45.54	37.40±28.15	
TG (g/l)	1.26±0.51	1.76±1.27	1.98±0.22	1.78±0.24	1
HDL-C (g/l)	0.46±0.12	0.51±0.34	0.50±0.29	0.50±0.002	
LDL (g/l)	1.14±0.36	1.09±0.43	1.23±0.14	1.27±0.11	1
TC (g/l)	0.31±0.02	1.91±0.72	2.31±0.07	1.92±0.17	1
GGT UI/L	45.53± 86.07	68.87±74.94	81.02±49.32	80.21±85.37	1
CRP mg/l	10.45±9.25	52.16±70.98	70.41±98.24	20.21±38.66	1
Albumin g/l	41.62±5.20	40.45±4.71	32.30±6.36	32.88±3.09	1

Table 1. Distribution of study patients according to NAFLD grades and the demographic, clinical and biochemical data

NAFLD ranks: grade 0: No FAFLD; grade 1: Steatosis; grade 2: Fibrosis; grade 3: Cirrhosis. BMI: body mass index. ASAT: Aspartate aminotransferase. ALAT: Alanine aminotransferase. TG: Triglycerides. TC: Total cholesterol. HDL: High density lipoprotein; LDL: low density lipoprotein. GGT: gamma-glutamyl transferase; CRP: C-reactive protein.

and GGT were significantly correlated with NAFLD grade 2 and 3. Table 3.

In addition, the multinomial log-probabilities of having steatosis and degrees 2 and 3 for people in the age category between 18 and 50 years compared to people aged \geq 50 years should decrease by 0.573 and

1.868 units respectively. In other words, people aged \geq 50 years are more likely to have NAFLD than people in the 18-50 age group. As for gender, men are more at risk of developing the advanced stages of steatosis than women (OR = 0.262; CI (0.071-0.96)

	<u> </u>		0					
BMI categories								
NAFLD grades	Normal weight N (%)	Overweight N (%)	Obesity N (%)	Total N (%)				
Grade 0	4(2.1)	9(4.7)	178(93.2)	191(100)				
Grade 1	10(6.1)	17(10.4)	137(83.5)	164(100)				
Grade 2	2(14.3)	2(14.3)	10(71.4)	14(100)				
Grade 3	6(35.3)	5(29.4)	6(35.3)	17(100)				
Total	22(5.7)	339 (8.5)	331 (85.8)	386(100)				

Table 2. Distribution of the study subjects according to their weight status and NAFLD grades

Grades of NAFLD: grade 0: No NAFLD; grade 1: Steatosis; grade 2: Fibrosis; grade 3: Cirrhosis

 Table 3. Summary of the multinomial logistic regression analysis results

NAELD	Variables	В	р	Exp(B)=OR	IC at 95 % for Exp(B)	
NALD	variables				lower bound	upper bound
Grade 1	CRP (mg/l)	0.049	0.000	1.05	1.031	1.07
	Age≥50 (between 18-50)	-0.573	0.038	0.564	0.328	0.97
	Triglycerides	1.005	0.000	2.733	1.752	4.264
Grade 2 &3 (fibrosis & cirrhosis)	CRP (mg/l)	0.047	0.000	1.048	1.028	1.069
	Triglycerides	1.11	0.004	3.036	1.433	6.432
	ALAT (UI/L)	0.068	0.000	1.07	1.034	1.108
	ALP UI/L	0.011	0.003	1.011	1.004	1.019
	GGT UI/L	-0.016	0.005	0.984	0.973	0.995
	Females vs males	-1.339	0.044	0.262	0.071	0.967
	Age≥50 (between 18-50)	-1.868	0.004	0.154	0.044	0.542
	BMI≥30 (between 18-25)	3.062	0.000	21.371	3.987	114.537
	BMI≥30 (between 25-30)	2.874	0.000	17.701	4.051	77.333

IC: intervalle de confiance ; OR: rapport de cotes. CRP:C reactive protein, ALAT: Alanine aminotransferase, ALP: Alkaline phosphatase, GGT: gamma-glutamyl transferase, BMI: body mass index

Models to predict NAFLD in the obese population

The prediction of NALFD and its severity is estimated, based on anthropometric and biological data from a sample of 386 Moroccans. Indeed, using the SPSS program, obesity is treated as a reference group and two models are estimated to predict steatosis and its advanced degrees, relative to obesity (Figure 1).

DISCUSSION

The results of the present study report an association between NAFLD and obesity. The prevalence of simple non-alcoholic fatty liver disease increased from 6.1% in people with a normal BMI to 10.4% in overweight people and to 83.5% in obese people.

```
ln(\pi 1/\pi 0) = -1.678 + 0.049 * CRP + 1.005 * TG - 0.573 * age
ln(\pi 2/\pi 0) = -5.356 + 0.047 * CRP + 1.110 * TG + 0.068 * ALAT + 0.011 * ALP - 0.016 * GGT - 1.339 * gender - 1.868 * age + 3.062 * BMI
```

ln: logarithm

 π 0: the probability of not having NAFLD π 1: the probability of having gradel NAFLD π 2: the probability of having grade 2-3 NAFLD

 $\pi 0 + \pi 1 + \pi 2 = 1$

Figure 1. Algorithm

The same is true for the severity and complications of NAFLD, which shows a prevalence of fibrosis of 14.3% in people of normal weight or overweight and 71.4% in obese. Cirrhosis was present in 35.3% of people with normal weight, 29% in overweight and 35.3% in obese. A significant association between BMI, waist circumference and NAFLD was also found. Consistent with these results, similar research in North America reported the presence of steatosis in 70% and NASH in 18.5% of obese people and severe fibrosis in 13.8% of obese patients [3]. Likewise, in the world population, the prevalence of NAFLD is estimated to be between 60% and 95% in obese people [19]. In addition, the global prevalence of obesity has been estimated at 51% among NAFLD patients and 81% in patients with NASH [2]. In addition, hyperlipidemia has been estimated to be 50% in the NAFLD population and has been observed as an increase in triglycerides and cholesterol in fatty liver disease [20]. In addition, another study stipulated that the global prevalence of non-alcoholic steatohepatitis in patients with type 2 diabetes was 37.3%. Also, among patients with NAFLD and type 2 diabetes who have had a liver biopsy, 17% have advanced fibrosis [21].

All these data converge towards a coexistence of NAFLD with obesity and the metabolic syndrome. However, NAFLD is not developed by all people with this syndrome, and vice versa [22, 23]. Comparably, data from the present study reports that 93.2% of the study sample were obese people but not all of them had NAFLD. Almost half of the obese (47.2%) had NAFLD and type 2 diabetes and just over half (52.8%) who had NAFLD but were not diabetic. On the other hand, while a statistically significant association was found between NAFLD and dyslipidemia, a proportion of 13.3% of this population had both NAFLD and dyslipidemia but in return 86.7% of patients with NAFLD did not have dyslipidemia. In addition, approximately 52% of subjects with NAFLD had hypertension, against 48% without hypertension but without revealing a statistically significant association between NAFLD and this disease.

Another determining factor in this study is age. This is because the prevalence of NAFLD increases with increasing age. People aged ≥ 50 are more likely to have steatosis and its advanced grades compared to younger people in the 18 and 50 age group. The multinomial log probabilities of having steatosis and grades 2 and 3 for people aged 18 to 50 compared to people ≥ 50 are expected to decrease by 0.573 and 1,868 units, respectively. This finding is consistent with data from several studies reporting that NAFLD occurs with increasing age[14, 20, 24]. Interestingly, both sexes could have NAFLD, but males were more likely than females to have fibrosis (grade 2). As in the present study, the prevalence of NAFLD in grades 1,

2 and 3 in females was 82.3%, 35.7%, 70.6% and that of males in the same grades was 17.7%, 64.3% and 29.4% respectively. These data are comparable to the results of several studies [14, 19, 24]. The multinomial log-probability of having advanced grades of steatosis for females was decreased by 1,339 units compared to males. One study reported that the relationship between gender and fibrosis may be influenced by menopausal status in women and that the incidence of NAFLD increases after the age of 50 with a peak at 60-69 years, and that NASH is more severe in women than in men [14, 25].

As for the levels of C-reactive protein and triglycerides, their elevations of one unit would respectively increase the log-probabilities of 0.049 and 1.005 for a person to have steatosis and would also respectively increase the log- probabilities of 0.047 and 1.110 for a person to have advanced degrees of NAFLD. In addition, raising the levels of alkaline phosphatase (ALP), alanine aminotransferase (ALT), and BMI by one unit would respectively increase the multinomial log-probabilities of 0.011, 0.068 and 3.062 for a person to have advanced grades of steatosis. These results are consistent with those in the literature which showed an increase in liver enzymes in 50% of patients with grade 1 NAFLD and in 80% in the advanced stages of the disease [22].

In sum, increased age, CRP and TG, gender, body mass index, ALAT, ALP, GGT are predictors of NAFLD and its severity. In this study, these factors were used to develop two algorithms to predict non-alcoholic fatty liver disease and its severity. Alternatively, these methods are rapid and avoid the risk of exposing the person to radiation and invasiveness of the biopsy as well as the relatively high expense of these diagnostic means.

CONCLUSION

Non-alcoholic fatty liver disease is a public health concern as it has become one of the most common liver diseases in the world due to its increasing incidence, largely explained by the increasing prevalence of obesity. The objective of the present study was to investigate the prevalence of non-alcoholic fatty liver disease linked to obesity in Morocco, to determine the associated risk factors as well as to develop a non-invasive procedure that can be assessed more objectively. The diagnosis of non-alcoholic fatty liver disease by biopsy is invasive and expensive, and there is substantial variability among observers in the ultrasound assessment of its severity. In this study two models were created that can be used to predict the degree of fat infiltration in NAFLD based on a person's demographic and biochemical data. They will help clinicians to easily diagnose this disease without the

need for specialized equipment or expertise. The two algorithms developed in this study need, however, to be validated by other studies and would be necessary to improve the quality of care for people with nonalcoholic fatty liver disease.

Acknowledgments

Thanks to the Director of CHU Med VI of Marrakech for authorizing us to consult the files and to the staff for their help. A special thanks to the patients for their participation, we wish them good recovery.

Conflict of interest

The authors declare that there is no conflict of interest.

REFERENCES

- García-Compeán D., Villarreal-Pérez JZ., Cavazos MEO., Lavalle-Gonzalez FJ., Borjas-Almaguer OD., Del Cueto-Aguilera AN., González-González JA., Treviño-Garza C., Huerta-Pérez L., Maldonado-Garza HJ.: Prevalence of liver fibrosis in an unselected general population with high prevalence of obesity and diabetes mellitus. Time for screening? Ann Hepatol 2020;19(3):258-264. doi: 10.1016/j.aohep.2020.01.003.
- Younossi ZM., Koenig AB., Abdelatif D., Fazel Y., Henry L., Wymer M.: Global epidemiology of nonalcoholic fatty liver disease-Meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology 2016;64(1):73-84 doi: 10.1002/hep.28431.
- Andronescu CI., Purcarea MR., Babes PA.: Nonalcoholic fatty liver disease: epidemiology, pathogenesis and therapeutic implications. J Med Life 2018;11(1):20-23.
- Castera L., Friedrich-Rust M., Loomba Castera R.: Noninvasive Assessment of Liver Disease in Patients With Nonalcoholic Fatty Liver Disease. Gastroenterology 2019;156(5):1264-1281 doi: 10.1053/ jgastro 2018; 12.036.
- S. Shannon A., Alkhouri N., Carter-Kent C., Monti L., Devito R., Lopez R., Feldstein AE., Nobili V.: Ultrasonographic quantitative estimation of hepatic steatosis in children With NAFLD. J Pediatr Gastroenterol Nutr 2011;53(2):190-5 doi: 10.1097/ MPG.0b013e31821b4b61
- Jennison E., Patel J., Scorletti E., Byrne CD.: Diagnosis and management of non-alcoholic fatty liver disease. Postgrad Med J. 2019;95(1124):314-322 doi: 10.1136/ postgradmedj-2018-136316.
- Loomba R., Cui J., Wolfson T., Haufe W., Hooker J., Szeverenyi N., Ang B., Bhatt A., Wang K., Aryafar H., Behling C., Valasek MA., Lin GY., Gamst A., Brenner DA., Yin M., Glaser KJ., Ehman RL., Sirlin CB.: Novel 3D Magnetic Resonance Elastography for the Noninvasive Diagnosis of Advanced Fibrosis in NAFLD: A Prospective Study. Am J Gastroenterol 2016;111(7):986-94 doi: 10.1038/ajg.2016.65.
- 8. Park CC., Nguyen P., Hernandez C., Bettencourt R., Ramirez K., Fortney L., Hooker J., Sy E., Savides MT., Alquiraish MH., Valasek MA., Rizo E., Richards L.,

Brenner D., Sirlin CB., Loomba R.: Magnetic Resonance Elastography vs Transient Elastography in Detection of Fibrosis and Noninvasive Measurement of Steatosis in Patients With Biopsy-Proven Nonalcoholic Fatty Liver Disease. Gastroenterology 2017;152(3):598-607 doi: 10.1053/j.gastro.2016.10.026.

- Godoy-Matos AF., Silva Júnior WS., Valerio CM. NAFLD as a continuum: from obesity to metabolic syndrome and diabetes. Diabetol Metab Syndr 2020;12:60 doi: 10.1186/s13098-020-00570-y.
- Pappachan JM., Babu S., Krishnan B., Ravindran NC. Non-alcoholic Fatty Liver Disease: A Clinical Update. J Clin Transl Hepatol 2017;5(4):384-393 doi: 10.14218/ JCTH.2017.00013.
- Anderson EL., Howe LD., Jones HE., Higgins JP., Lawlor DA., Fraser A. The Prevalence of Non-Alcoholic Fatty Liver Disease in Children and Adolescents: A Systematic Review and Meta-Analysis. PLoS One 2015;10(10):e0140908; doi: 10.1371/journal. pone.0140908.
- 12. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organization. Tech Rep Ser. 2000; 894: i-xii., 1-253.
- World Health Organization. [Report of the National Survey on Common Risk Factors for Non-Communicable Diseases Morocco (2017 – 2018)]. 2019.
- Habiba L., Mohamed M., Rekia., B.: Non-alcoholic fatty liver disease in Morocco: The situation., the determinants and the challenges for health care. World Journal of Advanced Research and Reviews 2020; 6(1)., 207-217doi: 10.30574/wjarr.2020.6.1.0100
- Belahsen R., Mziwira M., Fertat F. Anthropometry of women of childbearing age in Morocco: body composition and prevalence of overweight and obesity. Public Health Nutr. 2004;7(4):523-30 doi: 10.1079/ PHN2003570.
- World Health Organization-International Society of Hypertension Guidelines for the Management of Hypertension. Guidelines Subcommittee. J Hypertens1999; 17(2):151-83.
- Anwar S., Rashid H., Aleem B., Moslhey G- J., Al Rashdi A- S.: Correlation between anthropometric measurements and hypertension in Oman. Age (years) 2020; 48(1).53. 42.36-0.97.
- American Diabetes Association. Executive summary: Standards of medical care in diabetes2012. Diabetes Care 2012; 35:S4-S10 doi: 10.2337/dc12-s004.
- 19. *Baudin., B.*: Biochemical exploration of the liver in 2017. Francophone Journal of Laboratories 2017(490):25-33.
- Younossi ZM.: Non-alcoholic fatty liver disease
 A global public health perspective. J Hepatol 2019;70(3):531-544; doi: 10.1016/j.jhep.2018.10.033.
- Dixon JB., Bhathal PS., O'Brien PE.: Nonalcoholic fatty liver disease: predictors of nonalcoholic steatohepatitis and liver fibrosis in the severely obese. Gastroenterology 2001; 121(1):91-100 doi: 10.1053/gast.2001.25540.
- 22. Vanni E., Bugianesi E., Kotronen A., De Minicis S., Yki-Järvinen H., Svegliati-Baroni G. From the metabolic syndrome to NAFLD or vice versa? Dig Liver Dis 2010;42(5):320-30. doi: 10.1016/j.dld.2010.01.016.

- 23. López-Velázquez JA., Silva-Vidal KV., Ponciano-Rodríguez G., Chávez-Tapia NC., Arrese M., Uribe M., Méndez-Sánchez N.: The prevalence of nonalcoholic fatty liver disease in the Americas. Ann Hepatol. 2014;13(2):166-78; https://doi.org/10.1016/S1665-2681(19)30879-8
- 24. Estes C., Anstee QM., Arias-Loste MT., Bantel H., Bellentani S., Caballeria J., Colombo M., Craxi A., Crespo J., Day CP., Eguchi Y., Geier A., Kondili LA., Kroy DC., Lazarus JV., Loomba R., Manns MP., Marchesini G., Nakajima A., Negro F., Petta S., Ratziu V., Romero-Gomez M., Sanyal A., Schattenberg JM., Tacke F., Tanaka J., Trautwein C., Wei L., Zeuzem S., Razavi H.:

Modeling NAFLD disease burden in China., France., Germany., Italy., Japan., Spain., United Kingdom., and United States for the period 2016-2030. J Hepatol 2018; 69(4):896-904 doi: 10.1016/j.jhep.2018.05.036.

25. Goossens N., Bellentani S., Cerny A., Dufour JF., Jornayvaz FR., Mertens J., Moriggia A., Muellhaupt B., Negro F., Razavi H., Semela D., Estes C.: Nonalcoholic fatty liver disease burden - Switzerland 2018-2030 Swiss Med Wkly 2019;149:w20152 doi: 10.4414/ smw.2019.20152.

Received: 02.04.2022 Accepted: 12.08.2022

This article is available in Open Access model and licensed under a Creative Commons Attribution-Non Commercial 3.0.Poland License (CC-BY-NC) available at: http://creativecommons.org/licenses/by-nc/3.0/pl/deed.en