Prevalence of and risk factors for non-alcoholic fatty liver disease (NAFLD) and fibrosis among young adults in Egypt

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ABSTRACT

Objective Limited literature has examined the epidemiology of non-alcoholic fatty liver disease (NAFLD) and fibrosis among young adults in Egypt, a country with one of the highest obesity rates globally. We assessed the prevalence of steatosis and fibrosis among college students in Egypt.

Design In this cross-sectional study, we recruited students unaware of having fatty liver via a call-for-participation at a private university in the Dakahilia governorate of Egypt. Primary outcomes were the prevalence of steatosis as determined by the controlled attenuation parameter component of transient elastography and fibrosis as determined by the liver stiffness measurement component of transient elastography. Secondary outcomes were clinical parameters and socioeconomic factors associated with the presence and severity of steatosis and fibrosis.

Results Of 132 participants evaluated for the study, 120 (91%) were included (median (IQR) age, 20 (19–21) years; 65 (54.2%) female). A total of 38 participants (31.6%) had steatosis, among whom 22 (57.9%) had S3 (severe) steatosis. There was a higher risk for steatosis in persons with overweight (adjusted OR 9.67, 95% CI (2.94 to 31.7, p<0.0001) and obesity (adjusted OR 13.87, 95% CI 4.41 to 43.6, p<0.0001) compared with lean persons. Moreover, higher level of parental education was associated with progressing steatosis stages (S1–S3). Six (5%) participants had transient elastography values equivalent to F2–F3 fibrosis (four with F2 fibrosis ≥7.9 kPa, and two with F3 fibrosis ≥8.8 kPa).

Conclusion In this cohort of college students in Egypt, around 1 in 3 had steatosis, and 1 in 20 had moderate-to- advanced fibrosis, an established risk factor for hepatic and extrahepatic morbidity and mortality. These data underscore the urgency to address the silent epidemic of NAFLD among young adults in the Middle East-North Africa region.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has become the most common chronic liver disease with an estimated global prevalence of 25% of adults. NAFLD is a spectrum that comprises two main histological phenotypes with varying prognoses: NAFL or simple steatosis and non-alcoholic steatohepatitis (NASH). The latter is an advanced inflammatory form of NAFLD that confers higher risk of fibrosis, end-stage liver disease and cardiovascular disease mortality. In western societies, NASH prevalence among adults is estimated to be around 3%–4%, with as high as 40% of the cases progressing to advanced liver fibrosis. NAFLD progression is closely related to insulin resistance, obesity and type 2 diabetes mellitus.
2 diabetes, and is influenced by environmental factors (e.g., dietary fructose and alcohol; dysbiosis) and genetic predisposition (e.g., PNPLA3 polymorphism). Nevertheless, the complex pathogenesis of NAFLD remains incompletely understood.

Egypt, a Middle Eastern country with a population of ~100 million, with 60% of them being younger than 30 years, is considered among the highest 10 nations in prevalence of obesity. Overall, the Middle East and North Africa (MENA) region has one of the highest NAFLD prevalence rates, which is estimated to affect 31.8% of all adults. Young adults are often overlooked under the presumption they are ‘healthy’; however, the presence of NAFLD among this population could represent a major public health issue and may become a significant burden to healthcare systems in this region. In Western countries, cohort studies using ultrasonography (US) and transient elastography estimated the prevalence of NAFLD among adolescents 17–18 years of age to range between 2.5% and 20.7%. Despite that liver biopsy is the gold-standard method to confirm the presence of NAFLD and hepatic fibrosis, its use for wide screening of NAFLD is practically impossible and carries unnecessary risks. On the other hand, transient elastography is a simple, safe and reliable tool to assess steatosis and liver fibrosis. Using US, one cross-sectional study in Egypt evaluated 46 young adult women with obesity and polycystic ovary syndrome and found that 52% of them have NAFLD. To date, no studies in Egypt evaluated the prevalence of NAFLD and fibrosis among young adults using a widely accepted and validated technique. In this study, we assessed the prevalence of steatosis and fibrosis in a self-selected cohort of college students in Egypt using transient elastography.

METHODS
Study design and participants
This is a cross-sectional clinical study in which asymptomatic students (median [IQR] age, 20 [19–21] years) from a single university in the Dakahlia governorate of Egypt were recruited to undergo blood sampling and transient elastography (FibroScan 502 Touch; Echosens, Paris, France) in December 2018 at the university’s internal medicine clinics. University-wide recruitment was conducted through flyers in collaboration with the university facilities, students union, youth care administration and community services administration. The study was conducted on two separate ‘health day events’ and all participants who attended and signed informed consent on the first day were evaluated for the study. No remuneration was provided. This study followed the Strengthening the Reporting of Observational Studies in Epidemiology reporting guideline.

Assessment of outcomes
Participants were asked to come after overnight fasting for blood withdraw to measure serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST); lipid profile; plasma glucose, A1C, serum creatinine and serology for viral hepatitis (hepatitis A, B and C). Transient elastography to evaluate steatosis and fibrosis was performed on a separate day, and participants were asked to fast for at least 3 hours before the test. To capture a controlled attenuation parameter (CAP) score and liver stiffness measurement (LSM), 10 valid scans per subject were needed. If a participant’s median LSM was >7.1 kPa, the IQR-to-median ratio had to be <30% for this measurement to be considered valid. We initially used the M probe unless prompted by the device to switch to XL probe, which occurred in 31 (26%) participants, all with body mass index (BMI) ≥30 kg/m². Per the manufacturer instructions, cut-off values were unified for the M probe and XL probe.

A meta-analysis of studies containing histology verified CAP data was used to determine CAP score cut-offs for each steatosis grade (S0–S3) to determine the presence of NAFLD. S0 was defined as a CAP <248 dB/m (≤10% steatosis); S1 as CAP of 248 to <268 dB/m (10% to ≤33% steatosis (mild)); S2 as CAP of 268 to <280 dB/m (33% to ≤66% steatosis (moderate)); and S3 as CAP ≥280 dB/m (≥66% steatosis (severe)).

Cut-off values for transient elastography related to the Meta-analysis of Histological Data in Viral Hepatitis (METAVIR) scoring system were used to determine fibrosis staging (F0–F4). For F0–F1, LSM <7.9 kPa; for F2, LSM of 7.9 to <8.8 kPa; for F3, LSM of 8.8 to ≤11.7 kPa; and for F4 LSM ≥11.7 kPa.

Complete medical history, family history and social history were taken. Exclusion criteria included: prior diagnosis of a secondary cause of fatty liver such as Wilson’s disease, autoimmune hepatitis, alpha-1 antitrypsin deficiency or any other known secondary causes of fatty liver (e.g., excessive alcohol consumption). Alcohol consumption was assessed through social history, and excessive alcohol consumption was defined as >21 standard drinks per week for men and >14 standard drinks per week for women, where a standard drink is any drink that contains 14g of pure alcohol. Socioeconomic and lifestyle data were captured and were expressed as dichotomous variables. BMI and waist-to-height ratio were calculated.

Primary outcomes were the prevalence of steatosis as determined by the CAP component of transient elastography and the prevalence of liver fibrosis as determined by the LSM component of transient elastography. Secondary outcomes were clinical parameters and socioeconomic factors associated with the presence and severity of steatosis and liver fibrosis.

Statistical analysis
Univariable linear regression models were used to examine differences across ordinal categories of steatosis and fibrosis stages. Explanatory variables of interest were BMI categories, waist-to-height ratio, socioeconomic status (parental-level of education and residency), smoking, fast food consumption, exercise habits and sex. We used bivariate logistic regression
RESULTS

Participants’ characteristics are shown in Table 1. Of the 132 participants who were recruited, 2 refused to participate for personal reasons, and 4 had insufficient data and were excluded from the study. Six participants had unreliable fibroscan measurements and were excluded from the analysis. No participants had positive serology for acute or chronic viral hepatitis or reported significant alcohol consumption. Eventually, 120 participants were included in the analysis. This cohort included 65 women and 55 men. After checking medical history, no participants reported having a diagnosis of Wilson’s disease, autoimmune hepatitis or alpha-1 antitrypsin deficiency or any other known secondary causes of fatty liver. One participant had a diagnosis of hypertension and no participants had history of pre-diabetes or diabetes.

Table 2 shows participants’ characteristics across different steatosis stages. Median CAP score was 211.4 dB/m with a range of 100 to 400 dB/m. Thirty-eight participants (31.6%) had S1 or greater steatosis among whom 22 (57.9%) had S3 (severe) steatosis. BMI was positively associated with increasing (S1–S3) steatosis stages (p<0.0001), as was the proportion of subjects with obesity; being highest (63.6%) in subjects with S3 steatosis (figure 1). Steatosis in participants with BMI <25 kg/m² (lean NAFLD) was present in six participants (5%). Waist-to-height ratio as a surrogate for adiposity was also positively associated with increasing steatosis stages (p<0.0001, table 2). Similarly, triglycerides were positively associated with increasing steatosis stages, whereas HDL showed an inverse association (p=0.002 and p<0.0001, respectively, table 2). A1C values trended higher with increasing steatosis stages; being at a median of 5.2% (range: 4.9%–5.6%) in participants with S3 steatosis (p=0.012, table 2). ALT, AST, total cholesterol, LDL-cholesterol, plasma glucose and serum creatinine all showed no association with steatosis stages. Among participants with steatosis, 31 (81.6%) of 38 had ALT levels ≤30 IU/L for men and ≤19 IU/L for women.

The proportion of parental attainment of college education was positively associated with steatosis stages being highest (100%) among participants with S2 and S3 steatosis (p=0.019, table 2). Residency (rural vs urban) was not associated with steatosis, neither was a positive history of diabetes among first-degree relatives. Similarly, there was no association between steatosis stage and certain self-reported lifestyle habits: smoking, fast food consumption and exercise (table 2).

On regression analyses, participants with a BMI in the overweight (25–29.9 Kg/m²) and obese (>30 Kg/m²) ranges had significantly higher risk of steatosis (adjusted OR: 9.67; 95% CI 2.94 to 31.7; and 13.87; 4.41 to 43.6, respectively, p<0.0001 for both), than those with a BMI in the lean (<25 Kg/m²) category, after adjusting for lifestyle (ie, smoking, exercise and fast food consumption) and parental education (table 3). Similarly, increased adiposity as calculated from the waist-to-height ratio was associated with a significantly higher risk of steatosis in adjusted models (table 3). On regression analysis, parental attainment of college education did not confer a higher risk of steatosis in this cohort (table 3).
Transient elastography values ranged from 2.8 to 8.8 kPa with a median of 5.1 kPa (IQR: 4.25–6). Six (5%) of participants had transient elastography values equivalent to METAVIR F2-F3 fibrosis (four with F2 fibrosis ($\geq 7.9$ kPa), and two with F3 fibrosis ($\geq 8.8$ kPa)). No participants had F4 fibrosis ($\geq 11.7$ kPa). Although CAP score values were numerically higher with increasing fibrosis stages, this association was not statistically significant ($p=0.2$; table 4). Similarly, BMI values appeared to trend upwards with increasing fibrosis stages, but this trend was non-significant ($p=0.22$; table 4). The proportion of overweight subjects was positively associated with increasing fibrosis stages being highest (100%) in subjects with F3 fibrosis ($p=0.014$; table 4). Although a BMI in the overweight range appeared to confer greater risk of fibrosis on unadjusted regression analyses, the association faded after adjusting for lifestyle (ie, smoking, exercise and fast food consumption) and parental education (online supplemental table 1). Obesity was not associated with increased risk of fibrosis in our cohort (online supplemental table 1). Triglycerides were positively associated with increasing fibrosis stages ($p=0.015$; table 4),

| Table 2 | Demographic and clinical exposures across steatosis grades |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| S0 (n=82; 68.4%) | S1 (n=9; 7.5%) | S2 (n=7; 5.8%) | S3 (n=22; 18.3%) | P value * |
| Sex | NA | NA | NA | NA | 0.81 |
| Female | 44 (53.7) | 6 (66.7) | 3 (42.9) | 12 (54.5) | NA |
| Male | 38 (46.3) | 3 (33.3) | 4 (57.1) | 10 (45.5) | NA |
| Parental education | NA | NA | NA | NA | 0.019 |
| Lower than college | 7 (8.5) | 3 (33.3) | 0 | 0 | NA |
| College education | 75 (91.5) | 6 (66.7) | 7 (100) | 22 (100) | NA |
| Residency | NA | NA | NA | NA | 0.64 |
| Rural | 19 (23.2) | 2 (22.2) | 2 (28.6) | 8 (36.4) | NA |
| Urban | 63 (76.8) | 7 (77.8) | 5 (71.4) | 14 (63.6) | NA |
| Smoker | NA | NA | NA | NA | 0.45 |
| Yes | 5 (6.1) | 0 | 1 (14.3) | 3 (13.6) | NA |
| No | 77 (93.9) | 9 (100) | 6 (85.7) | 19 (86.4) | NA |
| BMI, kg/m² | 23.1 (19.5–27.2) | 26.1 (23.2–26.6) | 25.6 (19.1–30.7) | 31.8 (27–36.3) | <0.0001 |
| Underweight (<20 kg/m²) | 27 (32.9) | 2 (22.2) | 2 (28.6) | 0 | <0.0001 |
| Normal weight (20 to <25 kg/m²) | 28 (34.2) | 1 (11.1) | 0 | 1 (4.6) | <0.0001 |
| Overweight (25 to <30 kg/m²) | 14 (17.1) | 4 (44.5) | 3 (42.8) | 7 (31.8) | <0.0001 |
| Obese (≥30 kg/m²) | 13 (15.8) | 2 (22.2) | 2 (28.6) | 14 (63.6) | <0.0001 |
| Waist-to-height ratio | 0.47 (0.43–0.51) | 0.51 (0.43–0.51) | 0.51 (0.43–0.53) | 0.58 (0.49–0.64) | <0.0001 |
| First-degree history of diabetes | NA | NA | NA | NA | 0.39 |
| Yes | 26 (31.7) | 1 (11.1) | 3 (42.9) | 9 (40.9) | NA |
| No | 56 (68.3) | 8 (88.9) | 4 (57.1) | 13 (59.1) | NA |
| Fast food consumption | NA | NA | NA | NA | 0.31 |
| Yes | 55 (67.1) | 6 (66.7) | 7 (100) | 14 (63.6) | NA |
| No | 27 (32.9) | 3 (33.3) | 0 (0) | 8 (36.4) | NA |
| Exercise | NA | NA | NA | NA | 0.74 |
| Yes | 26 (31.7) | 2 (22.2) | 2 (28.6) | 9 (40.9) | NA |
| No | 56 (68.3) | 7 (77.8) | 5 (71.4) | 13 (59.1) | NA |
| Alanine aminotransferase, IU/L | 15 (11–21) | 15 (13–15) | 15 (12–24) | 15.5 (13–22) | 0.35 |
| Aspartate aminotransferase, IU/L | 21 (18–25) | 22 (19–24) | 21 (19–26) | 20.5 (17–23) | 0.29 |
| Total cholesterol, mg/dL | 166 (151–182) | 150 (139–173) | 171 (153–188) | 162 (151–179) | 0.86 |
| Triglycerides, mg/dL | 76 (59–94) | 67 (59–89) | 75 (68–92) | 106 (80–151) | 0.002 |
| LDL-C, mg/dL | 89 (75–105) | 85 (76–93) | 106 (83–116) | 91 (77–104) | 0.67 |
| HDL-C, mg/dL | 57 (51–64) | 51 (47–56) | 49 (46–54) | 50 (41–57) | <0.0001 |
| Plasma glucose, mg/dL | 102 (92–110) | 98 (91–106) | 99 (92–108) | 103 (94–110) | 0.92 |
| Glycated haemoglobin A1c, % | 5.1 (4.9–5.3) | 5 (5–5.1) | 5.1 (5–5.2) | 5.2 (5.1–5.4) | 0.012 |
| Serum creatinine, mg/dL | 0.99 (0.2) | 0.95 (0.2) | 0.95 (0.2) | 0.93 (0.2) | 0.63 |

Data are n (%), median (IQR) for non-normally distributed data or mean (SD) for normally distributed data as determined by Shapiro-Wilk test.

*Pearson’s χ² test, Wilcoxon rank-sum test or one-way analysis of variance (ANOVA).

BMI, body mass index; HDL-C, High-density lipoprotein; LDL-C, Low-density lipoprotein; NA, not applicable.
In our cohort of Egyptian college students, around 1 in 3 had steatosis and 1 in 20 had fibrosis. To the best of our knowledge, this is the first attempt to evaluate the prevalence of NAFLD among young Egyptian adults. It is known that the MENA region has one of the highest prevalence rates of NAFLD globally, and that Egypt ranked among the highest 10 nations that assessed 4021 young adults (median age 24 years) for hepatic steatosis and liver fibrosis using CAP. Knowing that NAFLD collectively included 5692 adults, 31.6% among young adults is exactly similar to the 31.8% prevalence rate that was shown in a meta-analysis of several epidemiological studies among populations that collectively included Middle Eastern populations. 1 

DISCUSSION

In our cohort of Egyptian college students, around 1 in 3 had steatosis and 1 in 20 had fibrosis. To the best of our knowledge, this is the first attempt to evaluate the prevalence of NAFLD among young Egyptian adults. It is known that the MENA region has one of the highest prevalence rates of NAFLD globally, and that Egypt ranked among the highest 10 nations that assessed 4021 young adults (median age 24 years) for hepatic steatosis and liver fibrosis using CAP. Knowing that NAFLD collectively included 5692 adults, 31.6% among young adults is exactly similar to the 31.8% prevalence rate that was shown in a meta-analysis of several epidemiological studies among populations. 1 

Table 3: Exposures associated with having steatosis

| Subjects with steatosis | Crude OR (95% CI) | P value | Adjusted for | Adjusted OR (95% CI) | P value |
|-------------------------|-------------------|---------|--------------|----------------------|---------|
| **BMI**                 |                   |         |              |                      |         |
| Lean*                   | 6/61 (9.8%)       | Reference | NA | Smoking, parental education, exercise, fast food | Reference | NA |
| Overweight              | 14/28 (50%)       | 9.16 (2.98 to 28.1) | <0.0001 | NA | 9.67 (2.94 to 31.7) | <0.0001 |
| Obese                   | 18/31 (58.1%)     | 12.7 (4.2 to 38.3) | <0.0001 | NA | 13.87 (4.41 to 43.6) | <0.0001 |
| Waist-to-height ratio   |                   | 0.64    |              |                      |         |
| Normal adiposity (<0.5)| 12/68 (17.6%)     | Reference | NA | Smoking, parental education, exercise, fast food | Reference | NA |
| Increased adiposity (>0.5)| 26/52 (50%)     | 4.66 (2.04 to 10.7) | <0.0001 | | 5.53 (2.3 to 13.3) | <0.0001 |
| **Parental education**  |                   |         |              |                      |         |
| School education        | 3/10 (30%)        | Reference | NA | BMI, smoking | Reference | NA |
| College education       | 35/110 (31.8%)    | 1.08 (0.3 to 4.4) | 0.9 | NA | 0.92 (0.2 to 4.2) | 0.9 |

*BMI <25 kg/m².

BMI, body mass index; NA, not applicable.
their study, 780 individuals (20.7%) were shown to have hepatic steatosis with 10% having S3 (severe) steatosis. This contrast may be attributed to the higher prevalence rates of age-standardised overweight and obesity in Egypt in comparison to UK (31.5% vs 26.1% for boys <20 years; and 39.5% vs 29.2% for girls <20 years, respectively). The prevalence of ≥F2 fibrosis in our cohort was approximately twice that reported in the UK study (5% vs 2.8%). Although transient elastography was extensively validated against liver biopsy, it has limited ability to discriminate between F0 and F1 fibrosis. This might lead to under-reporting of hepatic fibrosis among participants in our study. Although a BMI in the overweight category was significantly associated with higher risk for fibrosis; this association became non-significant after adjusting for other confounders.

While parental attainment of college education did not confer higher risk of steatosis on regression analyses, it
was proportionally higher with increasing steatosis stages (table 2). A study conducted in rural India reported that patients with NAFLD were more likely to have higher income. It also showed that higher family income is an independent predictor of NAFLD. Another study found that patients with NAFLD were more likely to live close to regions with abundant food resources (ie, grocery stores and restaurants). Western-style fast food chains that frequently serve calorie-dense meals are often accessible to higher-income families in Egypt and MENA region which contrasts with western societies where fast-food restaurants are widespread within low-income areas. In our cohort, self-reported lifestyle habits (ie, tendency to exercise, consumption of fast food and smoking) and other surrogates of socioeconomic status (ie, rural vs urban living) showed no association with steatosis. Meanwhile, findings from a large multicentre study among Hispanic/Latino individuals that examined the role of socioeconomic and lifestyle factors found no association with risk of developing NAFLD. We found obesity to be the strongest factor associated with progression of steatosis. On the other side of the spectrum, lean NAFLD was present in only 6 (5%) of participants.

In our cohort, increasing CAP scores were not associated with fibrosis progression. The trajectory of NAFLD progression from NAFL to NASH and fibrosis is nonlinear. Moreover, some patients may progress from steatosis to NASH fibrosis and even experience periods of spontaneous fibrosis regression on liver histology. Since our study was cross-sectional in design, it is plausible to expect that our reported fibrosis prevalence is underestimated.

We found no participants in our cohort to have A1C levels consistent with pre-diabetes or diabetes diagnosis, however, higher A1C levels were associated with progressing steatosis stages (S1–S3), which further supports the strong relationship between deteriorating glycaemia and the development of steatosis. Our observation is also in line with a recent report from China, which found that higher A1C within the non-diabetic range to be associated with incident NAFLD. Interestingly, 31 (81.6%) of 38 participants with steatosis and all 6 (100%) with ≥F2 fibrosis had normal ALT levels (≤30 IU/L for men and ≤19 IU/L for women). This observation underscores the poor utility of ALT alone as an initial screening test for NAFLD and fibrosis.

Our study had limitations. First, participants were recruited from a single university which may limit the study’s external validity and may introduce bias because responders to recruitment may have more concerns about their liver health than non-responders. However, the validated and rigorous methods of data collection allow for generalisability to the entire university. Second, comprehensive serological screening for secondary causes of fatty liver (ie, Wilson’s disease, autoimmune hepatitis or alpha-1 antitrypsin deficiency) was not done; however, serological screening was done for viral hepatitis and participants did not report having a diagnosis of any of the former conditions. Third, transient elastography has limited ability to distinguish between early fibrosis stages (F0 and F1); however, it has been extensively validated to detect significant fibrosis (≥F2). Therefore, our estimation of fibrosis prevalence should be interpreted with caution.

In conclusion, this study found steatosis is present in about one-third of an asymptomatic young adult cohort in Egypt. NAFLD confers increased risk of diabetes, cardiovascular disease, liver cirrhosis and cancer. At least 1 in 20 young adults had moderate-to-advanced fibrosis (F2–F3), which is concerning knowing that fibrosis is the most important predictor of morbidity and mortality in NASH. Although a minority of patients (3%–4% in western cohorts) will progress to NASH, those who do so could experience a fibrosis progression rate that ranges from 7 to 14 years per fibrosis stage. Higher socioeconomic status was associated with increased severity of steatosis in this cohort. This study serves as a call-to-action for Egyptian health authorities to curb the potential adverse implications of the obesity epidemic among young adults in Egypt. Furthermore, it provides rationale for conducting validation research on the population level to inform policy and public health interventions and increase awareness of NAFLD and its complications in Egypt and the MENA region.

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