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Liver Stiffness Values in Persons with Normal Histology

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Background and aims: **Most studies to date have focused on liver stiffness measurement (LSM) in patients with different chronic liver diseases, and normal LSM is defined based on normal liver function tests or the absence of fibrosis. Very few studies have defined LSM based on completely normal liver biopsies. The current study was done to define the distribution of LSM values in individuals with normal liver biopsies.** ***Methods:*** **All prospective liver donors presenting to Medanta, the Medicity hospital between September 2020 and September 2021 fulfilling the eligibility criteria were included in this study.** ***Results:*** **A total of 63 donors (36 females and 27 males) were included in the study, 37 (58.7%) donors had normal liver biopsies, and 26 (41.2%) donors showed the presence of non-alcoholic fatty liver disease. LSM values in the normal liver histology group were 5.01 ± 1.99 kPa by the M probe and 5.34 ± 2.25 kPa by the XL probe. Even though the correlation was weak (r = 0.29, *P* = 0.03), M probe LSM correlated positively with body mass index. There was a good correlation between the LSM measured by the M probe and the XL probe (r = 0.73, *P* = <0.001).** ***Conclusions:*** **LSM value in the biopsy-proven normal liver histology group was 5.01 ± 1.99 by the M probe and 5.34 ± 2.25 by the XL probe. (J Clin Exp Hepatol xxxx;xxx:xxx)**

Chronic liver injury due to any cause leads to fibrosis of the liver and may lead to cirrhosis. In some diseases like chronic hepatitis B and non-alcoholic fatty liver disease (NAFLD), the decision for pharmacological treatment also depends on the fibrosis stage. It is particularly true for patients with NAFLD, which is very common; only a few develop cirrhosis and progression takes many years. A liver biopsy is the gold standard to diagnose fibrosis but needs admission and carries the risk of complications that can be life-threatening.[1] There are several tests to measure fibrosis in a non-invasive way. Vibration-controlled transient elastography (TE) by FibroScan is a widely used and accepted modality to access fibrosis.[2–4] A meta-analysis showed that TE performed better than other non-invasive markers of fibrosis. Although magnetic resonance (MR) elastography was the best modality, its application is limited by availability.[5] Even though many studies have correlated the accuracy of TE with liver histology, almost all studies are in patients with chronic liver disease (chronic viral hepatitis, NAFLD, etc.) and only a few studies have compared the utility of TE and liver histology in determining the normal cut-offs for LSM in subjects with normal histology.[6–8] The obvious reason for the lack of literature in this area is that patients presumed to have normal histology would not be subjected to liver biopsy. Living donors being evaluated for liver transplantation provide an opportunity to biopsy such healthy subjects.

Prospective liver donors undergo a liver biopsy for various reasons like a low remnant, low graft-to-recipient weight ratio, and the presence of metabolic risk factors. Thus, prospective liver donors with normal liver biopsies are ideal to find out normal LSM. Only a few studies have looked at normal LSM values compared to normal liver histology in donors.[6–8] Our institute is a high-volume living donor liver transplantation (LDLT) center, thus the availability of donor liver biopsy provides an ideal opportunity to study LSM in persons with normal liver biopsy and no other disease. Hence, the primary aim of this study was to define the normal range of LSM values using TE in individuals with normal liver histology as determined by liver biopsy during evaluation as candidate donors for LDLT.

METHODOLOGY

The study was conducted at a tertiary care center in north India from September 2020 to September 2021. The study was approved by the institute's ethical committee and all participants gave informed consent. We aimed to study

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Abbreviations: LDLT: living donor liver transplantation; LSM: liver stiffness measurement; NAFLD: non-alcoholic fatty liver disease; TE: transient elastography
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normal LSM values in liver donors with normal liver histology. The donors with NAFLD on liver biopsy were taken as a comparative group. All candidate liver donors for LDLT who needed a liver biopsy were also subjected to a FibroScan examination. For the estimation of LSM with a mean of 4.3 and standard deviation of 1.2 (based on the previous study[8]), the sample size with 95% confidence worked out as 35. The inclusion criteria for the study included prospective liver donors, who had cleared all other stages of donor work up including clearances by cardiology, psychiatry, and pulmonary medicine. Prospective donors aged 21–57 years and body mass index (BMI) ranged from 20 to 32 kg/m^2. All donors underwent standard evaluation including negative viral markers (HBsAg, anti-HCV, and HIV) and the absence of significant alcohol intake (>21 units/week in males and >14 units/week in females). Following were the indications for liver biopsy: ≥2 metabolic risk factors (n = 42, 66.6%), <35% remnant liver volume or graft-to-recipient weight ratio <0.8 (n = 16, 25.3%), the suggestion of steatosis on non-contrast computed tomography (CT, <5 difference of average liver and splenic attenuation measures at multiple sites, n = 5, 7.9%). National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) criteria modified for Asians were used to diagnose metabolic syndrome.[9]

Vibration-controlled TE was assessed (by FibroScan 502 device EchoSens, Paris) in a fasting state of >4 h. A valid examination was defined as ≥10 valid LSM measurements with an interquartile range (IQR) interval/median ratio <30% and a success rate of >60%.

Statistical Methods

Data are shown as numbers and proportions for categorical variables. Parametric variables are shown as mean and standard deviation, and non-normal distributed quantitative variables are shown as median and 25–75 IQR. The study population was divided into 2 groups on the basis of histology, normal histology and ≥5% steatosis (grouped as NAFLD). Categorical outcomes were compared between normal histology and NAFLD groups using the Chi-square test. For non-normally distributed continuous parameters, medians and IQR were compared between study groups using the Mann–Whitney U test (2 groups). Association between quantitative explanatory and outcome variables was calculated by Pearson's correlation coefficient.

RESULTS

A total of 63 subjects were evaluated for inclusion in the study (27 [42.86%] males and 36 [57.14%] females). Out of 63 subjects, 26 (41.27%) participants had NAFLD. The hepatic steatosis on histology ranged from 5% to 25%; none of the subjects had histological evidence of non-alcoholic steatohepatitis. The NAFLD activity score was 2 or 3 in 4 patients (15.3%), 5 (19.2%) donors had evidence of ballooning of hepatocytes and 9 (34.6%) donors showed lobular inflammation. Stage 1 hepatic fibrosis was present in only 3 (11.5%) subjects and none had stage 2 or higher fibrosis. The baseline parameters between the 2 groups are shown in Table 1. There was a significant difference in alanine transaminase and controlled attenuation parameter (CAP) values between NAFLD and the normal histology group.

Metabolic risk factors and the presence of metabolic syndrome were higher in the NAFLD group; however, it did not reach statistical significance (Table 2).

All donors had reliable LSM values by either the M probe or XL probe, or with both probes, none had a failure of obtaining results by both the M and XL probes. Reliable

Table 1 Comparison of Baseline Parameters Between Normal Histology Versus NAFLD Groups.

Parameters	NAFLD (n = 26)	Normal histology (n = 37)	*P* value
Age (years)	35 (29–41)	36 (28–45)	0.28
Male gender	15 (57.69%)	12 (32.43%)	0.07
BMI (body mass index) (kg/m^2)	26.6 ± 3.25	25.7 ± 3.4	0.25
Waist circumference (cm)	92 ± 8.0	90.5 ± 9.43	0.49
Platelet count (10^3/dl)	241 ± 73	257 ± 89	0.45
International normalized ratio	1.01 ± 0.03	1.01 ± 0.047	0.62
Total bilirubin (mg/dl)	0.50 (0.4–0.8)	0.50 (0.4–0.6)	0.19
AST (aspartate transaminase) (IU/L)	29.50 (26.25–35.75)	27 (24–37)	0.11
ALT (alanine transaminase) (IU/L)	35.50 (26.5–40)	23 (17–40)	0.01
ALP (alkaline phosphatase) (IU/L)	76.50 (62.25–88)	82 (72–99)	0.12
GGT (Gamma-glutamyl Transferase) (IU/L)	24 (18–41.5)	24 (18–37)	0.50
LSM kPa	4.8 ± 0.97	5.1 ± 2.0	0.533
CAP db/m	264.3 ± 47.5	235.0 ± 50.5	0.024

Abbreviations: CAP, controlled attenuation parameter; LSM, liver stiffness measurement; NAFLD, non-alcoholic fatty liver disease.

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Table 2 Comparison of Metabolic Syndrome Risk Factors Among Subjects With Normal Histology and NAFLD.

Parameters	NAFLD (n = 26)	Normal histology (n = 37)	*P* value
Triglyceride (mg/dl)	154 ± 67	134 ± 52	0.18
High density lipoprotein (mg/dl)	41.65 ± 9.91	45.65 ± 11.30	0.15
Low density lipoprotein (mg/dl)	114.65 ± 36.9	100.11 ± 31.9	0.10
Fasting blood sugar (mg/dl)	95.85 ± 16.6	96.68 ± 11.75	0.81
Fasting blood sugar >100 (mg/dl)	1 (3.85%)	1 (2.70%)	1.0
Hypertension	2 (7.69%)	2 (5.41%)	1.0
HDL (mg/dl) <40 in males, <50 in females	18 (69.23%)	23 (62.16%)	0.56
Triglycerides (mg/dl) ≥150	11 (42.31%)	12 (32.43%)	0.42
Waist circumference (cm) ≥90 in males, ≥80 in females	22 (84.62%)	24 (64.86%)	0.08
Metabolic risk factors 0,1,2,3,4,5	1,6,10,8,0,1	4,10,17,5,1,0	0.87
Metabolic syndrome	9 (34.6%)	6 (16.6%)	0.13

Abbreviations: HDL, High density lipoprotein; NAFLD, non-alcoholic fatty liver disease.

readings on FibroScan could not be obtained in 7 of 63 donors with M probe (failure rate 11.1%) and 5 of 63 donors with XL probe (failure rate 7.9%). The BMI values were significantly higher in patients with M probe failure (28.5 ± 2.3 versus 25.7 ± 3.2, $P = 0.036$).

Nine patients had BMI >30 kg/m^2 in the study population. A total of 15 patients (24%) had a BMI of less than 23 kg/m^2. Four of these 15 patients were diagnosed as NAFL on biopsy, and 11 out of 15 patients had no NAFLD. There was a weak positive correlation between the LSM value by M probe and BMI (Pearson correlation coefficient 0.29, $P = 0.03$). There was no significant correlation between BMI and LSM values by XL probe (Pearson correlation coefficient 0.08, $P = 0.54$).

In 56 of 63 subjects (88.8%) in whom reliable readings were obtained with both the M and XL probe, there was a good correlation between the two probes [Pearson correlation of 0.73 ($P < 0.001$)]. Similarly, there was a good correlation between M and XL probes for measuring CAP values (Pearson correlation of 0.71 ($P < 0.001$).

DISCUSSION

The current study was done to define the normal distribution of LSM values in individuals with normal liver biopsy, which were 5.01 ± 1.99 by M probe and 5.34 ± 2.25 by XL probe. The use of LSM as a screening tool for liver disease in the general population is a promising idea, but normal LSM values have not been identified. As stated earlier, only a few biopsy-based studies have reported LSM in healthy persons. Fung *et al.* studied the LSM values of 28 healthy donors. The authors found that the median liver stiffness in healthy donors was 4.6 kPa, the liver stiffness value.[6] The study also included an occult hepatitis B cohort (n = 18) and none of the persons with LSM <7.2 kPa had fibrosis on liver biopsy.[6] Kim *et al.* showed that LSM values were 4.6 ± 0.5 kPa (range 3.3–5.6 kPa, 5th' and 95th percentiles being 3.9 and 5.3 kPa) in the healthy living liver and kidney donors in a cohort of 69 donors. Age and gender did not affect LSM values.[7] Alsebaey *et al.* found that median LSM was 4.3 ± 1.2 kPa in 50 liver donors. LSM values increased with BMI, donors with a BMI <26 kg/m^2 had significantly lower LSM values than donors having a BMI ≥26 kg/m^2 (4.0 ± 1.1 kPa versus 4.6 ± 1.2 kPa).[8] In the only study from India (liver biopsy not available for all), Das *et al.* noted higher LSM values at the higher or lower spectrum of BMI, which were significantly higher than subjects with normal BMI.[10] We also found that BMI correlated with LSM values by the M probe.

NAFLD is commonly associated with obesity but can occur in lean patients also.[11,12] NAFLD is very common in India with a prevalence in urban Indians ranging from 16% to 53%.[13–16] The prevalence is higher in patients with type 2 diabetes.[17,18] All these studies have used imaging to diagnose NAFLD, however, imaging may miss mild steatosis and the actual prevalence of NAFLD may be higher. A study from our center on healthy living liver donors found a 50.4% prevalence of NAFLD on biopsy.[19] Although, it can be argued that biopsy was done in selective donors with suspicion of NAFLD, a further study of the lean donor group (BMI < 23 kg/m^2) with none or only one metabolic risk factor still showed NAFLD in 28.5% of donors.[12] The current study differs from other studies as it was done in a preselected cohort of donors [age 18–55 years, non-morbidly obese, normal liver enzymes, and no gross steatosis as detected on CT (exclusion

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of donors with liver attenuation index values in minus)]. Histology in these subjects also showed a lower range of hepatic steatosis ranging from 5 to 25% making the prevalence of NAFLD 41.2%. Being a very specialized group, this cohort does not represent the general population. However, our data highlight the underestimation of NAFLD by most of the earlier studies which were based on ultrasound. As donors are preselected group (not diabetic, not morbidly obese, near normal liver enzymes), none of these subjects had evidence of NASH or significant fibrosis on histology.

Patients with NAFLD have metabolic syndrome more commonly than patients without NAFLD. There was no significant difference in the incidence of metabolic risk factors between the normal histology liver group and the NAFLD group in the current study. Although there was a trend of more metabolic syndrome in the NAFLD group, it did not reach statistical significance. As stated earlier, the preselected donor population is unlikely to have metabolic syndrome which should be the reason of less prevalence of metabolic syndrome in the current study. For the same reason, just 3 of 63 biopsies showed fibrosis. This is different from earlier Indian studies which showed NASH and fibrosis in a significant number of patients.[20–22]

A meta-analysis by Pu *et al.* showed an excellent area under the receiver operating characteristics (AUROC) (0.96) of CAP for diagnosing >5% steatosis.[23] Ultrasound is the most common modality used to diagnose NAFLD in clinical studies. The fact that CAP is good to diagnose mild steatosis (versus no steatosis) provides a superiority to CAP over ultrasound which may miss mild steatosis. A study from our center found that a CAP value of 262 dB/m was associated with good sensitivity and specificity (90% and 91%, respectively) to detect magnetic resonance imaging proton density fat fraction (MRI-PDFF) ≥5%.[24] In the current study cohort, we got lower cut-offs for CAP in comparison to what is described in the literature, even lower than a recent MR-PDFF based study from our center.[24] As other studies have included patients with all grades of steatosis (grade 1 to 3), those studies have found a higher CAP diagnostic cut-off as CAP values will be higher in grade 2 and 3 steatosis which results in a higher median value of CAP. The subjects with NAFLD in the current study had mild steatosis only (5–25%) due to predefined donor criteria. The donor selection criteria exclude donors with moderate or severe steatosis as we use a CT-measured liver attenuation index before doing a biopsy. Donors with liver attenuation index values in minus are asked for exercise and weight loss and are taken up for further evaluation only after reassessment on CT. So, we found lower CAP values that are not applicable to the general population but should be useful in ruling out steatosis.

The current study did not get any significant difference between M probe and XL probe values, and there was a good positive (r = 0.73) correlation between the M probe and XL probe regarding both CAP and LSM measurement. Thus, the XL probe may be used if results are not obtained by the use of the M probe, although it is not ideal as the correlation is not very strong.

The current study shows that there was a weak but statistically significant positive correlation between LSM value and BMI by the M probe. It was not true with the XL probe. Thus, the probe should be selected according to BMI. Less failure rate of the XL probe is consistent with the literature.[25]

The strengths of this study include a prospective cohort and the use of biopsy-proven normal histology as a reference standard. This is the first study of such kind from India. The present study is important because it spells out normal LSM values in the Indian population which could guide therapeutic decisions in patients with various liver diseases like NAFLD and hepatitis B where treatments are partly determined by the degree of fibrosis. The limitations of the study include being a single-center study with data mainly from North India. Also, as the study cohort was small, some outliers may have modified the results. The indications of liver biopsy and the limits of BMI and age may have modified results in the study groups; however, a universal biopsy of donors is neither possible nor ethical. To establish a normal range for a very common disease, it may not be a sufficient sample size with a small margin of error and this study can be considered a preliminary report. Larger sample size may also allow the assessment of the effect of increasing degrees of steatosis, inflammation, and early fibrosis on LSM values. A larger multicenter study would, therefore, be a welcome addition to our current understanding of normal LSM in the Indian population.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

DS, NSC, RS: Conceptualization; writing - original draft.
DS, SW, DG: writing draft, data collection.
AD, NS, ASS: critical revision.

CONFLICTS OF INTEREST

All authors have none to declare.

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SUPPLEMENTARY DATA

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