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Original Research

Relation between cardiac conduction disturbances and non-alcoholic fatty liver disease in the south Indian population

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Background: Non-alcoholic fatty liver disease (NAFLD) is known to increase the risk of cardiovascular diseases, including coronary artery disease and structural cardiac defects. Studies have suggested an association between NAFLD and atrial fibrillation, particularly in patients with advanced NAFLD and liver fibrosis. This study aimed to explore the potential link between NAFLD and cardiac conduction disturbances (CCDs), focusing on arrhythmias and other related factors.

Methods: This observational study was conducted over three months at a tertiary care hospital in Southern India. Participants were selected based on their health screening results and divided into two groups: Group 1 (NAFLD) and Group 2 (Non-NAFLD). All participants underwent ultrasound for NAFLD diagnosis and 12-lead ECG to assess cardiac rhythm abnormalities. Data on demographics, comorbidities, body mass index (BMI), lipid profiles, and other relevant factors were collected. Frequencies and types of CCDs were compared between the two groups.

Results: In a study of 178 patients undergoing preventive health screening, 105 (59.0%) were diagnosed with NAFLD. The mean age was 44.9±12.7 years, with 59.6% being male. Patients with NAFLD were older (46.5±11.6 vs. 42.6±13.8 years) and had higher rates of overweight BMI (66.7% vs. 42.5%, p=0.003) and arterial hypertension (31.4% vs. 17.8%, p=0.041) compared to those without NAFLD. No significant differences were found in the frequencies of diabetes mellitus or impaired glucose tolerance. Blood tests showed elevated serum glutamate pyruvate transaminase levels in the NAFLD group (36.37±28.04 vs. 28.23±21.28 IU/L, p=0.038). CCDs occurred in eight patients (4.5%), with no significant difference between NAFLD and non-NAFLD groups (4.8% vs. 4.1%, p=1.000). Logistic regression analysis showed no significant association between NAFLD and CCDs, both in univariate analysis (odds-ratio (OR)=0.86, 95% confidence interval (CI): 0.20-3.70, p=0.836) and multivariable analysis (OR=0.96, 95%CI: 0.20-4.70, p=0.962).

Conclusion: NAFLD, in the absence of significant cardiac comorbidities, does not appear to be associated with CCDs. These findings suggest that routine electrocardiographic screening for conduction abnormalities in asymptomatic NAFLD patients without pre-existing cardiac conditions may not be clinically justified. Healthcare resources should focus on comprehensive cardiovascular risk management and established metabolic risk factors rather than routine cardiac rhythm monitoring in this population. However, vigilance should be maintained for patients with advanced NAFLD or significant comorbidities.

Keywords: *Atrial Fibrillation; Arrhythmias, Cardiac; NAFLD; RBBB*

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1. Introduction

Non-alcoholic fatty liver disease (NAFLD) is an emerging, non-communicable disease which affects 25-30 % of the general population.1 Its prevalence rises to 70-75% among diabetics, while in obese people it rises to as high as 95-99%.1 In India, 10-30% of the population suffer from NAFLD and the number is higher among diabetics and obese patients.1 Each year, there are approximately 10 million reported cases of NAFLD in India.1 The lifestyle of people is increasingly sedentary with minimum exercise and consumption of food which is rich in fat and low in fibre thus leading to many complications.2 These two factors together have contributed to the rapid increase in the number of NAFLD cases.2–4 Research data has shown that people suffering from NAFLD are at an increased risk of developing cardiovascular complications like coronary artery disease (CAD) and cardiac structural defects.2–4

Studies have explored the association between NAFLD and atrial fibrillation (AF) with conflicting results.5–10 Ultrasound-verified prospective studies concur that NAFLD is associated with the incidence of AF.5–10 According to epidemiological evidence, the greater the prevalence of NALFD in a population, the stronger is its association with AF incidence and prevalence.5–10 Specifically, diabetic individuals with NAFLD are at the greatest risk of AF.9,11 Additionally, the risk of AF may concentrate most in individuals with advanced NAFLD, particularly those with liver fibrosis.5–10 The possible mechanistic links between NALFD and AF are diverse, with obesity and systemic inflammation having a significant role,11–15 but further studies are needed until NAFLD can be established as a causal factor in the incidence of AF.11–15 Some research evidence also shows the presence of other arrhythmic patterns including right bundle branch block (RBBB), but the studies to support that data are limited.11–15

Both NAFLD and arrhythmias are known to arise from excess of fat deposition in different organs.16 Therefore, it is important to study if there exists any relation between the fat deposition in the liver and the heart. If there exists any relation between the two, the fat deposition in the liver, causing NAFLD can act as a marker for deposition in the heart and can therefore help in early diagnosis of any possible arrhythmias. This study aimed to confirm the relation between NAFLD and cardiac conduction defects (CCDs). If the relation existed, we sought to evaluate the nature of the relation or the type of arrhythmia most commonly observed, as well as other factors that may have made NAFLD patients more prone to such complications. The hypothesis was that NAFLD patients have a higher frequency of CCDs and specific types of arrhythmias compared to those without NAFLD, potentially due to metabolic and inflammatory processes associated with the disease.

2. Methods

2.1. Study design and sample size calculation

This observational study was performed over a period of three months (July-September, 2019) at a tertiary healthcare centre in the Southern part of India, after obtaining approval from the Institutional Ethics Committee (IRB#CSP/19/JUN/78/233).

The sample size for this study was calculated to ensure sufficient power to detect a statistically significant association between the variables of interest. The calculation was done by a biostatistician with a confidence interval of 95%, a 5% margin of error and a target power of 80% to minimize the risk of Type II error.

2.2. Study population

The study population consisted of patients visiting the hospital for preventive health screening. Based on presence or absence of NAFLD, participants were divided into Group I (NAFLD) and Group II (Non-NAFLD). Initially 257 participants were selected by simple random sampling from the people who visited the preventive health screening at the tertiary care hospital. This was accomplished by randomly selecting outpatient files for all patients who visited the clinic on a specific day of the study, conducted without any consideration of patient information and in the absence of the patients themselves. Files of patients with incomplete

information were excluded from final analysis. Further, participants were excluded if their alcohol intake was greater than 20 g/day, had any pre-existing cardiac illness other than arterial hypertension or showed a liver disease other than NAFLD.

2.3. Experimental Design

After applying the inclusion criteria, 178 patients were included in the study and were grouped based on the presence of the disease as Group I and Group II **(Figure 1).**

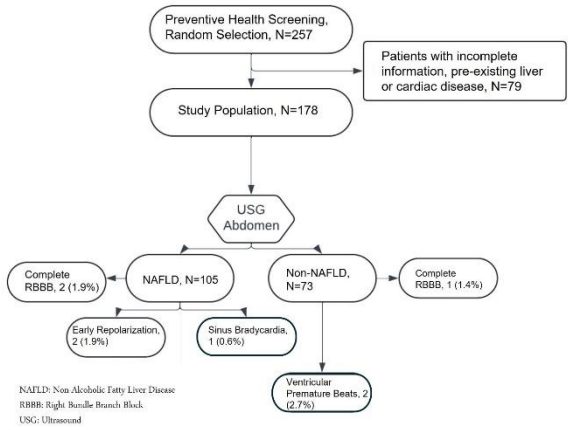

Figure 1. Flowchart summarizing the study protocol.

Patients of Group I were defined as those satisfying the following:

i. Ultrasound features assessed with 2-4 MHz vector transducer or 2-5 MHz convex transducer consistent with fatty liver.17 Absence of other causes of chronic liver disease [Hepatitis B Virus, Hepatitis C Virus), autoimmune hepatitis, toxins including alcohol (< 20 g/day)
ii. Absence of cirrhosis by imaging
iii. Absence of pre-existing cardiac conditions other than arterial hypertension

Participants of Group II were those satisfying the following:

i. Ultrasound showing no features of fatty liver
ii. No evidence of liver disease
iii. No cardiac illness other than arterial hypertension

NAFLD was further graded based on the ultrasound findings:

Grade 1: Echogenicity of the liver is increased;

Grade 2: Echogenic liver obscures the echogenic walls of portal vein branches, and,

Grade 3: Echogenic liver obscures the diaphragmatic outline.[18]

After obtaining written informed consent from the patients, they were asked about their medical history, alcohol intake and family medical history. At presentation, basic patient information were collected (including age, sex, body mass index (BMI), whether they have diabetes mellitus or arterial hypertension, and serum lipid levels).

Patients were subjected to routine blood tests including glycaemia (mg/dL), liver function tests [ie; total bilirubin (mg/dL), alkaline phosphate (IU/L), albumin (g/dL), serum glutamic oxaloacetic transaminase (SGOT, IU/L) and serum glutamate pyruvate transaminase (SGPT, IU/L)], lipid profile [ie; total cholesterol (mg/dL), triglycerides (mg/dL), low density lipoprotein (LDL, mg/dL) and high density lipoprotein (HDL, mg/dL)], and renal function tests [ie; serum creatine (mg/dL)]. Impaired glucose tolerance was defined as serum glucose levels between 110-126 mg/dL.

All patients underwent ultrasound scanning of the abdomen. The diagnosis of fatty liver was based on the ultrasound as per criteria mentioned earlier.17 Any other abnormality in the liver or any other abdominal organ was also noted. The ultrasound was also used to grade the level of fatty liver as described earlier.17 Further, all the patients were subjected to a standard 12-lead electrocardiogram (ECG) to record their cardiac rhythm. Based on the data collected, the frequency of

CCDs in people suffering from NAFLD was calculated. The ECGs of both the groups were studied in detail by an experienced internal medicine consultant, abnormalities were noted, and further the results of the two groups were compared in order to understand the role of NAFLD in causing the abnormality.

The frequency of NAFLD among various groups and against various characteristics like age, sex, BMI, whether they have diabetes mellitus, lipid levels, were studied in detail to find out the demographic characteristics of our participant group and therefore interpret the results obtained accordingly. The BMI was classified into underweight (<18.5 kg/m2), normal weight (18.5-24.9 kg/m2), overweight (25.0-29.9 kg/m2) and obese (>30.0 kg/m2). [19]

3. Statistical Analysis

Descriptive statistics were employed to summarize the demographic data of the participants. Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as means with standard deviations (SD). After assessing the data for normal distribution, a two-tailed t-test was conducted for continuous variables. For categorical variables Chi-square test (X2) or Fisher's exact test were utilized based on the sample size. Binary logistic regression was also performed. A significance level (α) of less than 0.05 was considered statistically significant. All statistical analyses were carried out using SPSS software.

4. Results

Of the 178 patients who underwent preventive health screening during the study period, 105 (59.0%) had NAFLD while 73 (41.0%) did not have NAFLD. The characteristics of the study population have been summarized in Table 1. Patients with NAFLD were older, had high rates of overweight BMI, and arterial hypertension as compared to those who did not have NAFLD. There was no significant difference in the rate of diabetes mellitus or impaired glucose tolerance between the two groups. Among the 105 patients with NAFLD, 83 (79.0%) had grade 1, 18 (17.1%) had grade 2, and 4 (3.8%) had grade 3 disease.

Blood tests performed during the preventive health screening revealed a higher SGPT level (36.37±28.04 vs. 28.23±21.28 IU/L, p=0.038) in patients with NAFLD as compared to those without NAFLD; however, no difference was seen in other parameters including haemoglobin, total bilirubin, SGOT, albumin and lipid or renal profile (all p>0.005) between the two groups.

CCDs were seen in 8 (4.5%) patients. There was no difference in the rate of CCDs between the two groups, with 5 in patients with NAFLD and 3 in patients without NAFLD; the difference was not statistically significant (4.8% vs. 4.1%, p=1.000). The most common CCD was complete RBBB (37.5%) followed by early repolarization (25.0%) and ventricular premature beats (25.0%) and only 1 (12.5%) patient had sinus bradycardia (Table 2). There was no association between the grade of NAFLD and CCDs (p=0.964).

On binary logistic regression, univariate analysis (odds-ratio (OR)=0.86, 95% confidence interval (CI): 0.20-3.70, p=0.836) or multivariable analysis controlling for age, sex, BMI, diabetes mellitus and arterial hypertension (OR=0.96 95%CI:0.20-4.70, p=0.962) did not reveal any significant relation between NAFLD and CCDs **(Table 3)**.

	Total (n=178)	**NAFLD (n=105)**	**Without NAFLD (n=73)**	**p-value**
Age (years), mean±SD	44.9±12.7	46.5±11.6	42.6±13.8	**0.045**
Sex, male, n (%)	106 (59.6%)	67 (63.8%)	39 (53.4%)	0.165
Body mass index (kg/m^2), n (%)				
Underweight: < 18.5	6 (3.4%)	2 (1.9%)	4 (5.5%)	**0.003**

Normal weight 18.5-24.99	63 (35.4%)	27 (25.7%)	36 (49.3%)	
Overweigh t 25-29.99	101 (56.7%)	70 (66.7%)	31 (42.5%)	
Obese: > 30	8 (4.5%)	6 (5.7%)	2 (2.7%)	
Diabetes mellitus, n (%)	37 (20.8%)	25 (23.8%)	12 (16.4%)	0.2 33
Impaired glucose tolerance, n (%)	30 (16.9%)	21 (20.0%)	9 (12.3%)	0.2 90
Arterial hypertension, n (%)	46 (25.8%)	33 (31.4%)	13 (17.8%)	**0.0 41**
Haemoglobin (g%), mean±SD	13.4±1.7	13.5±1.6	13.1±2. 0	0.3 46
Serum creatinine (mg/dL), mean±SD	0.9±0.8	0.9±1.0	0.8±0.7	0.5 95
Liver function				
Total bilirubin (mg/dL), mean±SD	0.8±0.4	0.8±0.4	0.8±0.3	0.5 55
Serum glutamate pyruvate transaminase (IU/L), mean±SD	33.0±25. 7	36.4±28. 0	28.2±2 1.3	**0.0 38**
Serum glutamic oxaloacetic transaminase(IU/L), mean±SD	29.0±17. 5	30.48±1 8.6	26.75± 15.7	0.1 63
Alkaline phosphate (IU/L), mean±SD	97.738.0	98.0±38. 1	97.37± 38.0	0.0 81
Albumin (g/dL), mean±SD	4.2±0,3	4.2±0.3	4.2±0.3	0.6 69
Lipid profile				
Total Cholesterol (mg/dL), mean±SD	182.7±4 2.5	183.6±4 3.2	181.3± 41.6	0.7 31
Triglycerides (mg/dL), mean±SD	149.0±1 05.9	158.3±1 16.2	135.5± 88.2	0.1 58
Low Density Lipoprotein (mg/dL), mean±SD	125.1±3 1.9	126.8±3 1.6	122.8± 32.4	0.4 22
High Density Lipoprotein (mg/dL), mean±SD	40.9±7.5	40.3±7.3	41.7±7. 7	0.0 99
Cardiac Conduction Disturbances, n (%)	8 (4.5%)	5 (4.8%)	3 (4.1%)	1.0 00

Table 1. Characteristics of study population and between group comparisons for presence of arrhythmia.

Conduction defect	**NAFL D (n=105)**	**NAFL D grade**	**Withou t NAFL D (n=73)**	**Total, N=8 (%)**
Complete Right Bundle Branch Block, n (%)	2 (1.9%)	Grade 1: 1 Grade 2: 1	1 (1.4)	3 (37.5%)
Early repolarisatio n, n (%)	2 (1.9%)	Grade 1: 2	0 (0.0%)	2 (25.0%)
Ventricular premature beats, n (%)	0 (0.0%)	-	2 (2.7%)	2 (25.0%)
Sinus bradycardia, n (%)	1 (0.6%)	Grade 1: 1	0 (0.0%)	1 (12.5%)

NAFLD: Non-Alcoholic Fatty Liver Disease.

Table 2. Frequency and types of arrhythmias among the study population.

Table 3. Univariate and multivariable logistic regression controlling for age, sex, body mass index, diabetes mellitus and arterial hypertension.

Outcome	Exposure	Univariate		Multivariable	
		OR (95% Confidence Interval)	P-value	OR (95% Confidence Interval)	P-value
Cardiac Conduction Disturbances	Non-Alcoholic Fatty Liver Disease	0.86 (0.20-3.70)	0.836	0.96 (0.20-4.70)	0.962

5. Discussion

The present study did not observe a statistically significant association between NAFLD and CCDs after analysing the data of 178 participants out of which 105 were NAFLD patients. The frequency of CCDs was 4.5% with no significant difference between patients with and without NAFLD. Among the NAFLD patients who developed a cardiac arrhythmia, complete RBBB (1.9%) and early repolarisation (1.9%) were the most CCDs observed.

Prior studies evaluating the relationship between NAFLD and CCDs, have found a higher arte of AF in NAFLD.5–10 Our data, however, differs from the already available results and concluded that the frequency of arrhythmias in NAFLD patients is statistically insignificant. The main reason for the low and insignificant frequency of CCDs among NAFLD patients is exclusion of patients with prior cardiac illness other than arterial hypertension unlike most prior studies, which have included patients with cardiac illness.5–10 Some of the participants in previous studies were on treatment with medications like digoxin and beta-blockers which itself can cause CCD.

In our study, the average age of participants was 45 years, while the average age of patients who had NAFLD was 46.5 years, which is significantly lower when compared to other studies. In a Finnish study, the average age of NAFLD patients was 63 years.6 In a Chinese study, participants were selectively aged > 65 years with the average age being 72 years.7 In an American study, while all patients were above 18 years, the average age of those with NAFLD was 59 years.9 In an Indian study, the ECGs of 400 asymptomatic people aged between 45 and 74 years were studied.12 This Indian study has outlined the overall relationship between the electrocardiographic abnormalities and advancing age. There exists a parallel relationship between the incidence of abnormal ECG and advancing age according to this study, as the age increases the number of abnormal ECGs increases.12 In one largest community based prospective Japanese study, which evaluated the relation between AF and age, a positive relationship was found, that is, age promotes the development of AF.13 Thus the data collected and analysed in previous studies may have been influenced by the older age of the participants, and therefore giving the results that might not be an accurate representation of the disease itself.

In our study, out of 178 participants studied only 26% had arterial hypertension, and out of the 105 patients with NAFLD, only 31% had arterial hypertension. It is known that hypertensives are more prone in developing arrhythmias as compared to non-hypertensives.20 Thus, with many previous studies, including a high number of hypertensives in their participant pool, could have altered the observation. In an American study, the participant group included 60% hypertensives while in those with NAFLD, 64% were hypertensives.5 A prior study evaluated the independent risk factors for AF in 4731 participants, and reported that arterial hypertension was an independent risk factor for AF (OR, 1.5 for males and 1.4 for females). Arterial hypertension alone accounted for 14% of the AF burden in both sexes, and was one of the independent predictors for new-onset AF.14 This aforementioned study highlighted the

role of arterial hypertension in causing AF, and therefore signifies how having large number of hypertensives among their participants could have altered the results of previous studies.14

In our study, participants with pre-existing cardiac disorders were specifically excluded to rule out any other abnormalities, which could cause an arrhythmia, but many other previous studies have included patients with CAD and congestive heart failure, which may be responsible for arrhythmias independently.5,15 In an American study, out of 408 NAFLD participants, 23 had history of heart failure, and 45 of CAD. In a Finnish study, out of 400 patients, 11 had heart failure, 19 CAD and 131 had cardiac artery stenosis.5 In a follow up Finnish study, 79 participants among a total 958 had history of CAD.6 Prior literature evaluating the independent risk factors for AF among 4731 participants, and congestive heart failure was found to be an independent risk factor for AF with OR 4.5 for males and 5.9 for females. The OR for myocardial infarction as an independent risk factor in the same study was 1.4.15 Thus, it is clear that the pre-existing cardiac conditions can cause arrhythmias especially AF, which was the main finding of many of previous studies.5,15

In some studies, the included participants were on long-term drug therapies. Certain drugs used by the participants pool are known to be arrhythmogenic.6 In a Finnish study, 53 participants were on calcium channel blockers, and 26 on beta-blockers. In an American study, 10.4% of participants were illicit drug users.6

In our study, ECGs were studied in detail, and the automated result was not used since they may show false QT prolongation when the T wave amplitude is small or when T wave is very flat. QTc was calculated manually using conventional formula which QT interval is observed divided by square root of R-R interval.21 This method showed a normal QT in all participants. Thus, all these factors together can alter the results obtained in various study and therefore this relationship need to be studied in detail before any strong conclusion is made. Our study provides research data from a demographically distinct group of participants andincluded a wide array of participants,which was not the case in the previous studies, where groups that are more specific were selected for research, which could alter their results.6,7,9 The ECG readings were corrected and read by a specialist, and the auto-generated readings were not used, therefore reducing the chances of error in reading.

There are certain limitations in our study. Based on the previous data available, the sample size we calculated was small because of the high frequency of the conduction defects, but our study showed a statistically insignificant relation between them and therefore our findings need to be confirmed with a larger study sample. Secondly, the study was conducted at a single institute in South India, and therefore, the results cannot be generalized. Multi-institutional studies with diverse populations should be undertaken to further confirm the findings of this study. Furthermore, although limiting our patient pool to only arterial hypertension as a cardiac disease and excluding all others decreased confounding, but limited the scope of the study. Another limitation in our study would be not following up with the NAFLD patients over the years but rather recording their ECG at a random time of their disease and studying it. There might be a chance that the patients might develop arrhythmias later in the course of their disease.

6. Conclusion

This study demonstrates that NAFLD, in the absence of significant cardiac comorbidities, does not independently increase the risk of cardiac conduction disturbances (CCDs). While NAFLD is strongly associated with metabolic dysfunction, systemic inflammation, and cardiovascular risk, these findings suggest that its impact on cardiac electrophysiology is limited in patients without advanced liver disease or significant cardiac conditions. These results highlight the need to focus on

managing broader cardiovascular risk factors in NAFLD patients rather than routine screening for CCDs in asymptomatic individuals. Future studies should explore this relationship in populations with advanced NAFLD or higher systemic inflammatory burden to better delineate the potential electrophysiological effects of liver disease progression.

ETHICAL APPROVAL

The study has been approved by Sri Ramachandra Institute of Higher Education & Research Ethics Committee for Student Projects

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CONFLICT OF INTEREST

The Authors declare that there is no conflict of interest

DATA AVAILABILITY STATEMENT

The datasets used and analysed during the current study are available from the corresponding author on reasonable request.

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REFERENCES

1. Perumpail BJ, Khan MA, Yoo ER, Cholankeril G, Kim D, Ahmed A. Clinical epidemiology and disease burden of nonalcoholic fatty liver disease. World J Gastroenterol. 2017;23(47):8263-8276. doi:10.3748/wjg.v23.i47.8263
2. Wong VWS, Wong GLH, Yip GWK, et al. Coronary artery disease and cardiovascular outcomes in patients with non-alcoholic fatty liver disease. Gut. 2011;60(12):1721-1727. doi:10.1136/gut.2011.242016
3. Patil R, Sood GK. Non-alcoholic fatty liver disease and cardiovascular risk. World Journal of Gastrointestinal Pathophysiology. 2017;8(2):51. doi:10.4291/wjgp.v8.i2.51
4. Mookadam F, Goel R, Alharthi MS, Jiamsripong P, Cha S. Epicardial fat and its association with cardiovascular risk: a cross-sectional observational study. Heart Views. 2010;11(3):103-108. doi:10.4103/1995-705X.76801
5. Mangi MA, Rehman H, Minhas AM, Rafique M, Bansal V, Constantin J. Non-Alcoholic Fatty Liver Disease Association with Cardiac Arrhythmias. Cureus. Published online April 13, 2017. doi:10.7759/cureus.1165
6. Käräjämäki AJ, Kettunen O, Lepojärvi S, et al. Presence of atrial fibrillation is associated with liver stiffness in an elderly Finnish population. PLoS ONE. 2017;12(3):e0173855. doi:10.1371/journal.pone.0173855
7. Zhang Y, Li P, Miao M, et al. Nonalcoholic Fatty Liver Disease Is Associated with Increased Atrial Fibrillation Risk in an Elderly Chinese Population: A Cross-Sectional Study. BioMed Research International. 2018;2018:1-7. doi:10.1155/2018/5628749
8. İşcen S. RBBB is associated with an increased risk of NAFLD in young healthy individuals. Int J Cardiol. 2013;168(4):4056-4057. doi:10.1016/j.ijcard.2013.07.035
9. Targher G, Valbusa F, Bonapace S, et al. Non-alcoholic fatty liver disease is associated with an increased incidence of atrial fibrillation in patients with type 2 diabetes. PLoS One. 2013;8(2):e57183. doi:10.1371/journal.pone.0057183
10. Long MT, Yin X, Larson MG, et al. Relations of Liver Fat With Prevalent and Incident Atrial Fibrillation in the Framingham Heart Study. JAHA. 2017;6(5):e005227. doi:10.1161/JAHA.116.005227
11. Gupta S, Gupta RK, Kulshrestha M, Chaudhary RR. Evaluation of ECG Abnormalities in Patients with Asymptomatic Type 2 Diabetes Mellitus. J Clin Diagn Res. 2017;11(4):OC39-OC41. doi:10.7860/JCDR/2017/24882.9740
12. Khane R, Surdi A, Bhatkar R. Changes in ECG pattern with advancing age. Journal of basic and clinical physiology and pharmacology. 2011;22:97-101. doi:10.1515/JBCPP.2011.017
13. Boriani G. Atrial Fibrillation and Aging: Risky Mutual Relationships. Chest. 2016;149(2):301-302. doi:10.1016/j.chest.2015.08.018
14. Verdecchia P, Angeli F, Reboldi G. Hypertension and Atrial Fibrillation: Doubts and Certainties From Basic and Clinical Studies. Circulation Research. 2018;122(2):352-368. doi:10.1161/CIRCRESAHA.117.311402
15. Benjamin EJ, Levy D, Vaziri SM, D'Agostino RB, Belanger AJ, Wolf PA. Independent risk factors for atrial fibrillation in a population-based cohort. The Framingham Heart Study. JAMA. 1994;271(11):840-844.
16. Galiero R, Caturano A, Vetrano E, et al. Pathophysiological mechanisms and clinical evidence of relationship between Nonalcoholic fatty liver disease (NAFLD) and cardiovascular disease. RCM. 2021;22(3):755-768. doi:10.31083/j.rcm2203082
17. Lăpădat AM, Jianu IR, Ungureanu BS, et al. Non-invasive imaging techniques in assessing non-alcoholic

fatty liver disease: a current status of available methods. Journal of Medicine and Life. 2017;10(1):19.
18. Saadeh S, Younossi ZM, Remer EM, et al. The utility of radiological imaging in nonalcoholic fatty liver disease. Gastroenterology. 2002;123(3):745-750. doi:10.1053/gast.2002.35354
19. CDC. Adult BMI Categories. BMI. July 1, 2024. Accessed January 5, 2025. https://www.cdc.gov/bmi/adult-calculator/bmi-categories.html
20. Afzal MR, Savona S, Mohamed O, Mohamed-Osman A, Kalbfleisch SJ. Hypertension and Arrhythmias. Heart Failure Clinics. 2019;15(4):543-550. doi:10.1016/j.hfc.2019.06.011
21. Al-Khatib SM, LaPointe NMA, Kramer JM, Califf RM. What Clinicians Should Know About the QT Interval. JAMA. 2003;289(16). doi:10.1001/jama.289.16.2120