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Research Article

# The Prevalence of Cardiovascular Diseases in Patients with Fatty Liver Disease and Study on the Correlation Between Fatty Liver Disease and Atrial Fibrillation

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## Abstract

*Objective:* This study aims to investigate the prevalence of cardiovascular diseases in patients with fatty liver disease (FLD) and explore the correlation between FLD and atrial fibrillation (AF). *Methods:* Participants with relatively complete clinical data were selected from the physical examination center of the First Affiliated Hospital of Soochow University from January 2019 to December 2021. The prevalence rates of cardiac conduction system lesions, and cardiac structural and functional changes in the group with fatty liver and group without fatty liver were analyzed and compared. The association between FLD and AF was proved by Logistic regression analysis. *Results:* For cardiac conduction system lesions, the prevalence of atrioventricular block, bundle branch block, and AF in the population with FLD was higher than those without fatty liver, while the prevalence of premature beats was slightly lower. There were statistical differences in the prevalence of atrioventricular block and AF between the two groups ($P < 0.001$). For changes in cardiac structure and function, the prevalence of atrial enlargement, ventricular hypertrophy, and cardiac insufficiency was higher in the fatty liver group than in the non-fatty liver group, while the prevalence of valvular reflux was slightly lower. There were statistical differences in the prevalence of atrial enlargement and ventricular hypertrophy between the two groups ($P < 0.001$). Univariate Logistic regression analysis demonstrated FLD was linked with the occurrence of AF (OR 1.767, 95% CI 1.439-2.168). After adjusting for confounding factors by multivariate Logistic regression analysis, the correlation between FLD and AF remained statistically significant (OR 1.993, 95% CI 1.199-3.313). *Conclusions:* Compared with those without fatty liver, the prevalence of atrioventricular block, AF, atrial enlargement, and ventricular hypertrophy in patients with FLD was higher and the differences were statistically significant. FLD was an independent risk factor for AF, which remained true after adjusting for multiple confounding factors.

## Keywords

Fatty Liver Disease, Cardiovascular Diseases, Atrial Fibrillation, Metabolic Syndrome, Insulin Resistance

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# 1. Introduction

In recent years, with changes in lifestyle and dietary structure, the prevalence of fatty liver disease (FLD) has been increasing annually and shows a trend towards affecting younger populations. According to statistics, the global prevalence of FLD is approximately 25.24%, with the highest rates observed in the Middle East and South America, and the lowest in Africa. The prevalence in Asia is approximately 27.37%, making it a global public health issue [1]. Several studies have shown a significant association between the presence and severity of FLD and the risk of coronary artery disease, arrhythmias (including various supraventricular and ventricular arrhythmias, as well as atrioventricular block), and structural and functional changes in the heart (such as valvular heart disease, ventricular hypertrophy, and cardiac dysfunction) [2-7]. As one of the common persistent arrhythmias, atrial fibrillation (AF) can lead to serious consequences such as stroke, heart failure, and sudden cardiac death, significantly impairing quality of life. With growing awareness of the cardiovascular risks associated with FLD, the potential link between FLD and AF has garnered particular attention. Currently, whether FLD independently contributes to the occurrence of AF remains a controversial topic, and further exploration of the relationship between the two is required.

# 2. Materials and Methods

## 2.1. Patients

Subjects with relatively complete physical examination data from the Physical Examination Center of the First Affiliated Hospital of Soochow University between January 2019 and December 2021 were selected. Based on abdominal ultrasound/CT findings, the subjects were divided into two groups: the FLD group and the non-FLD group. From 2019 to 2021, the FLD group included 39,160 individuals who completed electrocardiogram (ECG) and 5,573 who completed cardiac ultrasound. In 2021, the non-FLD group included 32,515 individuals who completed ECG and 3,827 who completed cardiac ultrasound. A total of 1,673 subjects were selected and grouped based on ECG results and past medical history. Among them, 769 cases diagnosed with AF constituted the case group, while 904 without AF served as the normal control group.

## 2.2. Clinical Data

Physical examination data including ECG, cardiac ultrasound and past medical history were collected for both the FLD and non-FLD group.

General clinical data of the AF group and the normal control group were collected and statistically analyzed, including gender, age, history of hypertension, history of diabetes, history of fatty liver, past medical history, body mass index (BMI), albumin (ALB), alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), left atrial diameter (LAD), and ejection fraction (EF).

## 2.3. Observation Indicators

(1) The prevalence rates of common cardiac conduction system disorders, as well as cardiac structural and functional changes, were statistically compared between the FLD and non-FLD group. (2) The general clinical data of the AF group and the normal control group were compared. Logistic regression analysis was used to analyze the correlation between FLD and AF.

## 2.4. Statistical Analysis

Microsoft Excel and SPSS 26.0 were used. For continuous data, a normality test was first conducted. If the data followed a normal or approximately normal distribution, they were expressed as mean ± standard deviation, and the t-test was used for intergroup comparison; if the data did not follow a normal distribution, they were expressed as median (P25~P75), and the Mann-Whitney U test was used for intergroup comparison. Categorical data were expressed as frequency and percentage, and intergroup comparisons were performed using the chi-square test. Correlation analysis was conducted using logistic regression analysis, calculating the odds ratio (OR) and its 95% confidence interval (CI). A P-value $< 0.05$ was considered statistically significant.

# 3. Results

## 3.1. Comparison of the Prevalence of Common Cardiac Conduction System Disorders Between the FLD Group and Non-FLD Group

As shown in Table 1 and Figure 1, the prevalence rates of atrioventricular block, left/right bundle branch block, and AF in the FLD group were all higher than those in the non-FLD group, while the prevalence of premature beats was similar between the two groups. The differences in the prevalence of atrioventricular block and AF were statistically significant ($P < 0.001$).

***Table 1.*** *Prevalence of Common Cardiac Conduction System Disorders in the FLD Group and non-FLD Group.*

| Cardiac conduction system disorders | FLD group n=39160 | non-FLD group n=32515 | *P* value |
|---|---|---|---|
| Atrioventricular block | 666 (1.70%) | 390 (1.20%) | <0.001 |
| Left/Right bundle branch block | 803 (2.05%) | 605 (1.86%) | 0.068 |
| AF | 313 (0.80%) | 156 (0.48%) | <0.001 |
| Premature beats | 689 (1.76%) | 577 (1.77%) | 0.928 |

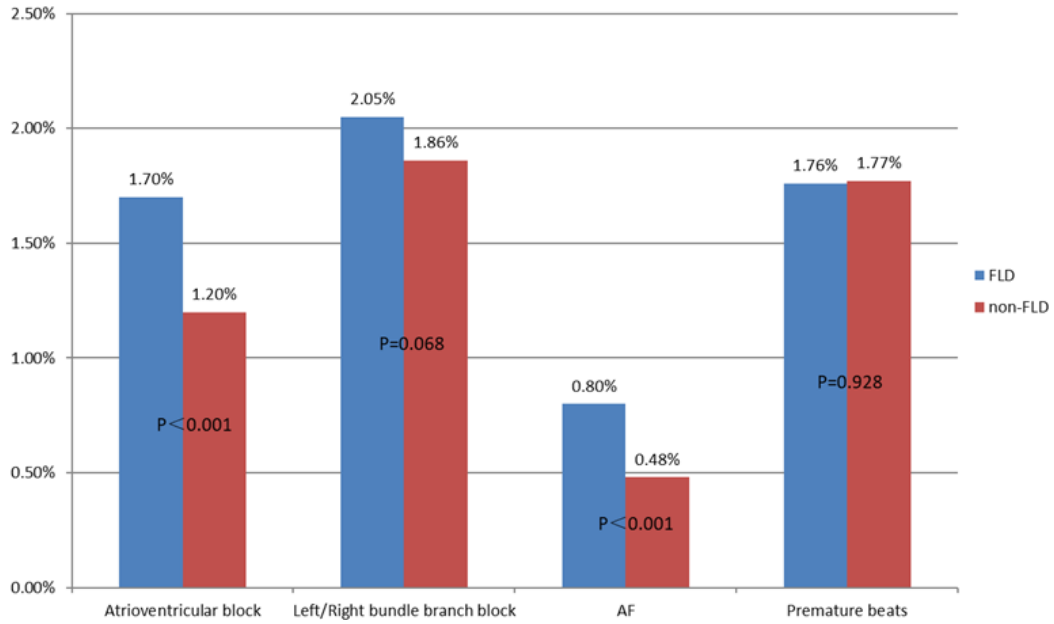


***Figure 1.*** *Prevalence of common cardiac conduction system Disorders in the FLD group and non-FLD group.*

## 3.2. Comparison of the Prevalence of Common Cardiac Structural and Functional Changes Between the FLD Group and Non-FLD Group

As shown in Table 2 and Figure 2, the prevalence rates of atrial enlargement, ventricular hypertrophy, and cardiac insufficiency in the FLD group were all higher than those in the non-FLD group, while the prevalence of cardiac valvular regurgitation was similar between the two groups. The differences in the prevalence of atrial enlargement and ventricular hypertrophy were statistically significant (P < 0.001).

***Table 2.*** *Prevalence of Common Cardiac Structural and Functional Changes in the FLD Group and non-FLD Group.*

| Cardiac structural and functional changes | FLD group n=5573 | non-FLD group n=3827 | *P* value |
|---|---|---|---|
| Atrial enlargement | 1023 (18.36%) | 570 (14.89%) | <0.001 |
| Cardiac valvular regurgitation | 774 (13.89%) | 572 (14.96%) | 0.150 |
| Ventricular hypertrophy | 753 (13.51%) | 230 (6.01%) | <0.001 |
| Cardiac insufficiency | 54 (0.97%) | 31 (0.81%) | 0.424 |

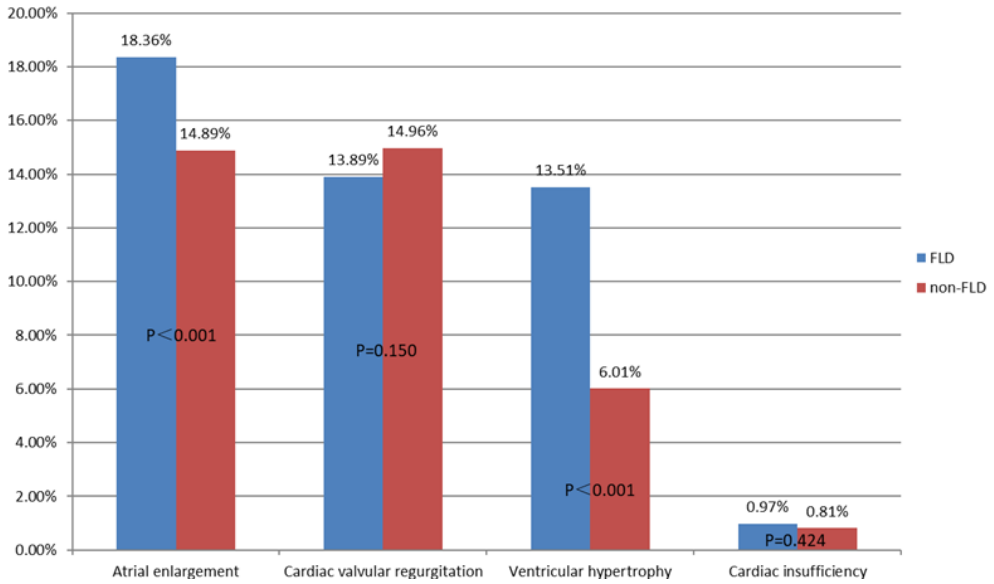


***Figure 2.*** *Prevalence of common cardiac structural and functional changes in the FLD group and non-FLD group.*

## 3.3. Comparison of Baseline Characteristics Between the AF Group and the Normal Control Group

There were statistically significant differences in general data such as gender, age, history of hypertension, history of diabetes, history of fatty liver, BMI, ALB, ALT, GGT, TC, LDL-C, HDL-C, LAD, and EF between the two groups ($P < 0.05$) (Table 3).

***Table 3.*** *Baseline Characteristics between the AF Group and the Normal Control Group.*

| | **AF group n=769** | **Normal control group n=904** | ***P* value** |
|---|---|---|---|
| Gender (%) | | | |
| male | 571 (74.25%) | 425 (47.01%) | <0.001 |
| female | 198 (25.75%) | 479 (52.99%) | |
| Age (year) | 71.10 ±13.24 | 42.54 ±12.41 | <0.001 |
| Hypertension (%) | | | |
| no | 277 (36.02%) | 699 (77.32%) | <0.001 |
| yes | 492 (63.98%) | 205 (22.68%) | |
| Diabetes (%) | | | |
| no | 527 (68.53%) | 801 (88.61%) | <0.001 |
| yes | 242 (31.47%) | 103 (11.39%) | |
| FLD (%) | | | |
| no | 458 (59.56%) | 653 (72.23%) | <0.001 |
| yes | 311 (40.44%) | 251 (27.77%) | |
| BMI ($kg/m^2$) | 25.64 ±3.57 | 24.31 ±3.44 | <0.001 |
| ALB (g/L) | 44.36 ±3.04 | 47.18 ±2.42 | <0.001 |
| ALT (U/L) | 19.50 (14.90, 26.65) | 18.50 (12.50, 30.08) | 0.003 |
| AST (U/L) | 22.50 (19.10, 27.40) | 21.40 (17.70, 26.10) | 0.737 |
| GGT (U/L) | 30.30 (20.25, 46.80) | 20.60 (15.00, 35.28) | 0.002 |

| | AF group n=769 | Normal control group n=904 | *P* value |
|---|---|---|---|
| TC (mmol/L) | 4.63±1.00 | 5.17±0.99 | <0.001 |
| TG (mmol/L) | 1.31 (0.94, 1.88) | 1.17 (0.80, 1.70) | 0.059 |
| LDL-C (mmol/L) | 2.67±0.90 | 3.14±0.91 | <0.001 |
| HDL-C (mmol/L) | 1.29±0.34 | 1.35±0.36 | <0.001 |
| LAD (mm) | 47.28±7.16 | 36.50±3.28 | <0.001 |
| EF (%) | 62.28±7.08 | 63.90±3.76 | <0.001 |

FLD: fatty liver disease, BMI: body mass index, ALB: albumin, ALT: alanine aminotransferase, AST: aspartate aminotransferase, GGT: gamma-glutamyl transferase, TC: total cholesterol, TG: triglyceride, LDL-C: low-density lipoprotein cholesterol, HDL-C: high-density lipoprotein cholesterol, LAD: left atrial diameter, EF: ejection fraction.

## 3.4. Association Between FLD and AF

In the unadjusted logistic regression model, there was an association between fatty liver and the occurrence of AF (OR 1.767, 95% CI 1.439-2.168). In Model 1, the association between fatty liver and AF remained statistically significant after adjusting for gender, age, hypertension, diabetes, and BMI. As shown in Model 2, further adjustment for ALB, TC, LDL-C, and HDL-C did not substantially alter this association. Model 3 additionally included the following factors: LAD, EF, ALT, and GGT. In this final model, fatty liver, gender, age, hypertension, ALB, LAD, and EF were independently associated with the occurrence of AF (Table 4).

***Table 4.*** *Association Between Fatty Liver and the Occurrence of AF.*

| Variables | Logistic regression model | | | |
|---|---|---|---|---|
| | **Unadjusted model OR (95%CI)** | **Adjusted model 1 OR (95%CI)** | **Adjusted model 2 OR (95%CI)** | **Adjusted model 3 OR (95%CI)** |
| Fatty liver | 1.767 (1.439-2.168) | 1.629 (1.142-2.325) | 2.035 (1.384-2.993) | 1.993 (1.199-3.313) |
| Gender | | 3.556 (2.505-5.047) | 3.532 (2.417-5.159) | 3.852 (2.305-6.436) |
| Age | | 1.154 (1.138-1.170) | 1.136 (1.119-1.153) | 1.108 (1.088-1.128) |
| Hypertension | | 1.732 (1.248-2.404) | 1.962 (1.389-2.771) | 1.990 (1.274-3.106) |
| Diabetes | | 0.844 (0.577-1.236) | 0.888 (0.595-1.325) | 1.074 (0.638-1.808) |
| BMI | | 1.013 (0.961-1.067) | 1.009 (0.955-1.066) | 0.938 (0.873-1.009) |
| ALB | | | 0.828 (0.772-0.888) | 0.831 (0.758-0.910) |
| TC | | | 0.594 (0.412-0.857) | 0.769 (0.477-1.240) |
| LDL-C | | | 1.129 (0.769-1.658) | 0.941 (0.570-1.554) |
| HDL-C | | | 1.224 (0.668-2.245) | 1.067 (0.478-2.382) |
| LAD | | | | 1.414 (1.342-1.489) |
| EF | | | | 0.939 (0.902-0.977) |
| ALT | | | | 0.987 (0.970-1.004) |
| GGT | | | | 1.000 (0.995-1.005) |

BMI: body mass index, ALB: albumin, TC: total cholesterol, LDL-C: low-density lipoprotein cholesterol, HDL-C: high-density lipoprotein cholesterol, LAD: left atrial diameter, EF: ejection fraction, ALT: alanine aminotransferase, GGT: gamma-glutamyl transferase. Note: Independent predictors of AF are highlighted in bold.

## 4. Discussion

With the increasing prevalence and hazards of FLD, it has now become a widely recognized health concern among the public [8]. Studies have found that the risk of cardiovascular disease (CVD) in patients with FLD is 64% higher than that in the non-FLD population, and this risk further increases when liver fibrosis is present [9]. On this basis, our study further investigated and compared the prevalence of common CVDs between the FLD and non-FLD populations. The results showed that the prevalence rates of atrioventricular block, AF, atrial enlargement, and ventricular hypertrophy in the fatty liver population were all statistically significant higher than those in the general population ($P < 0.001$), which is consistent with the findings of several previous studies as shown below [10-13, 15-18].

FLD has adverse impacts on the anatomical structure of the heart. Research has found that compared with patients with hypertension alone, those with both FLD and hypertension have an enlarged LAD, suggesting that fatty liver may exacerbate structural changes in the left atrium, mainly manifested as left atrial enlargement [12]. Hallswort et al. [13] demonstrated that compared with the healthy population, the changes in the cardiac structure of patients with FLD are significant, mainly manifested as left ventricular hypertrophy, and there is a correlation between FLD and left ventricular hypertrophy.

In addition, in recent years, it has been recognized that FLD also affects the cardiac electrophysiological system. Research indicates that individuals with type 2 diabetes and FLD have a threefold higher risk of developing persistent cardiac conduction block compared with those without FLD [10]. AF is one of the most common arrhythmias. While past treatments primarily focused on anticoagulation and ventricular rate control, current research proposes incorporating risk factor management into the treatment strategy [14]. An increasing number of studies have explored the relationship between FLD and AF. Most studies have shown that FLD is independently associated with the occurrence of AF [15-18]. A prospective study spanning 16.3 years showed that 14.9% of patients with FLD were diagnosed with AF, compared to only 7.9% in those without FLD. After adjusting for confounding factors such as age, gender, diabetes, BMI, waist circumference, liver enzymes, left ventricular mass index, LAD, atrial natriuretic peptide, and high-sensitivity C-reactive protein, it was found that FLD independently increased the risk of AF regardless of these risk factors [15]. In addition, in a 10-year follow-up study of 400 patients with type 2 diabetes, Targher et al. [18] found that the risk of new-onset AF in patients with FLD was higher than that in those without FLD, and this correlation was independent of age, gender, hypertension, and ECG characteristics (left ventricular hypertrophy and PR interval).

There are several potential mechanisms linking FLD to AF. Currently, fatty liver tends to be regarded as a pathological syndrome of insulin resistance (IR), chronic low-grade inflammation, hypercoagulability, and ectopic fat deposition [19]. Firstly, fatty liver may exacerbate IR, leading to the release of various pro-inflammatory, pro-fibrotic, and vasoactive mediators, which can cause alterations in cardiac rhythm. These changes may worsen with the progression of liver disease [20]. In the state of IR, cardiomyocytes take up free fatty acids exceeding their oxidative capacity. Excess free fatty acids form triglycerides, and the excessive deposition of fatty acids and triglycerides in cardiomyocytes can induce cardiac lipotoxicity, impairing ventricular or atrial diastolic function and thereby triggering AF [21]. Additionally, IR can cause endothelial cell dysfunction and increase platelet aggregation rate. Meanwhile, the liver is a major source of prothrombotic molecules, increasing the production of fibrinogen and coagulation factor VIII, further elevating the risk of thrombosis [22]. Some studies have found that AF patients exhibit a hypercoagulable state, which seems to hint at a connection between the two [23]. Secondly, the activation of the nuclear factor kappa-B pathway in fatty liver promotes the transcription of various pro-inflammatory cytokines, leading to the massive secretion of inflammatory cytokines and inducing oxidative stress [24]. Previous studies have shown that levels of inflammatory biomarkers such as high-sensitivity C-reactive protein and interleukin-6 in the AF population are higher than in the general population, suggesting that chronic inflammation is an important risk factor for AF [25]. Finally, some studies have reported that fatty liver is an independent risk factor for autonomic nervous system dysfunction [26], which is itself a risk factor for AF [27]. This may also partly explain the connection between fatty liver and AF.

Research in the field of the correlation between FLD and AF is promising, and prospective studies are needed in the future for further confirmation. The possible pathogenic mechanisms of the association between the two provide new therapeutic targets for preventing and treating electrophysiological changes in cardiomyocytes in patients with FLD. This could help reduce the substantial medical and economic burden caused by cardiovascular complications in these patients.

## 5. Conclusions

Individuals with FLD have been shown to have higher prevalence rates of atrioventricular block, AF, atrial enlargement, and ventricular hypertrophy compared with those without FLD, with statistically significant differences. FLD is confirmed as an independent risk factor for the occurrence of AF, and this association remains true after adjusting for multiple confounding factors.

## Abbreviations

| | |
|---|---|
| FLD | Fatty Liver Disease |
| AF | Atrial Fibrillation |
| ECG | Electrocardiogram |
| BMI | Body Mass Index |
| ALB | Albumin |
| ALT | Alanine Aminotransferase |
| AST | Aspartate Aminotransferase |
| GGT | Gamma-glutamyl Transferase |
| TC | Total Cholesterol |
| TG | Triglyceride |
| LDL-C | Low-density Lipoprotein Cholesterol |
| HDL-C | High-density Lipoprotein Cholesterol |
| LAD | Left Atrial Diameter |
| EF | Ejection Fraction |
| OR | Odds Ratio |
| CI | Confidence Interval |
| CVD | Cardiovascular Disease |
| IR | Insulin Resistance |

## Author Contributions

**Xiaolan Shi:** Conceptualization, Investigation, Funding acquisition, Writing-review & editing.

**Yijie Gu:** Data curation, Formal Analysis, Supervision.

**Su Yan:** Project administration, Resources, Software, Validation.

## Ethics Approval and Consent to Participate

The study was approved by the Medical Ethics Committee of the First Affiliated Hospital of Soochow University (Approval No. 2022-327).

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## Conflicts of Interest

The authors declare no conflicts of interest.

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