New-onset Atrial Fibrillation is Associated With Polycystic Kidney Disease
A Nationwide Population-based Cohort Study

Tung-Min Yu, MD, Ya-Wen Chuang, MD, Mei-Ching Yu, PhD, Shih-Ting Huang, MD, Che-Yi Chou, PhD, Cheng-Li Lin, MSc, Chun-Ching Chiu, MD, and Chia-Hung Kao, MD

Abstract: Cardiovascular complications remain the major problems contributing to morbidity and mortality in patients with polycystic kidney disease (PKD).

Therefore, the authors hypothesized that atrial fibrillation (AF) is closely associated with PKD. The authors conducted a nationwide population-based cohort study to investigate the risk of AF in patients with PKD.

Using data from inpatient claims, the authors enrolled 7203 patients aged over 20 years who were diagnosed with PKD from 1998 to 2010 with no history of AF as the PKD cohort. They randomly selected 28,739 people without PKD as controls and frequency matched them with patients with PKD according to their age, sex, and baseline comorbidity.

In total, 247 PKD patients were diagnosed with AF, representing an incidence of 7.08 per 1000 person-years, whereas 807 cases of AF occurred in the comparison cohort, yielding an incidence of 4.98 per 1000 person-year, with an adjusted HR (aHR) of 1.31 (95% CI = 1.14–1.51).

The risk of AF increased from an aHR of 1.59 (95% CI = 1.15–2.21) to 3.64 (95% CI = 1.93–6.85) when the number of risk factors increased from 1 to more than 5 in comparison with patients without risk factors.

A remarkably high incidence rate and risk was observed in patients with PKD when multiple risk factors were combined. A high index of suspicion should be maintained when examining PKD patients with irregular betas. Early prophylactic therapy is warranted in these patients.

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Abbreviations: AF = atrial fibrillation, aHR = adjusted hazard ratio, BNHI = Bureau of National Health Insurance, CI = confidence interval, LHID 2000 = Longitudinal Health Insurance Database 2000, NHI = National Health Insurance, NHIA = National Health Insurance Research Database, NHIRD = National Health Insurance Research Database, NHR = National Health Research Institutes, PKD = polycystic kidney disease.

INTRODUCTION

Atrial fibrillation (AF) is the most clinically prevalent arrhythmia and frequently associates with severe cardiovascular complications. Atrial arrhythmogenic remodeling has been suggested to play a fundamental role in mediating the development of atrial arrhythmia, which refers to any change in cardiac diseases and conditions. The mechanisms involved in AF are complex and include structural remodeling, autonomic nervous system changes, and abnormal changes in Ca2+ handling.

First, structural remodeling presents as atrial enlargement; tissue fibrosis has been assumed to be closely associated with the development of persistent AF; and atrial dimensions have been assumed as a key determinant of persistent AF. Second, the activation of the autonomic nervous system is suggested to be closely associated with the initiation and maintenance of AF, and adrenergic activation may enhance L-type, ryanodine receptor 2 subtype (RyR2) open probability, and SR Ca2+ load. Finally, accumulating evidence has indicated that intracellular Ca2+ metabolic disorders may contribute to the induction and activation of proarrhythmia remodeling through Ca2+ cell-related signal pathways.

Polycystic kidney disease (PKD) is a prevalent genetic disorder and autosomal dominant polycystic kidney disease (ADPKD) is the most common disease, occurring in approximately 1 in 500 people. Mutations of PKD1 account for 85% of ADPKD cases, and mutations of PKD2 account for 15%; PKD1 and PKD2 encode the proteins polycystin-1 (PC1) and polycystin-2 (PC2), respectively. Although PKD typically manifests with renal cysts, extrarenal manifestations, particularly in cardiovascular abnormalities, are occasionally observed. Cardiovascular complications remain the major problems contributing to morbidity and mortality in patients with PKD. Left
VENTRICULAR HYPERTRPHY, MITRAL VALVE PROLAPSE, AND AORTIC ANEURYSM ARE OCCASIONALLY OBSERVED IN PATIENTS WITH PKD.\(^\text{10–13}\) ACCUMULATING EVIDENCE HAS REVEALED THAT THE ENLARGEMENT OF RENAL CYSTS INHERENTLY ACTIVATES THE RENIN–ANGIOTENSIN–ALDOSTERONE SYSTEM (RAASS) AND SYMPATHETIC ACTIVITY IN BOTH CIRCULATING AND INTRA-RENAL CYST.\(^\text{14}\) A PREVIOUS STUDY IDENTIFIED ALL OF THE MAJOR COMPONENTS OF THE RENIN–ANGIOTENSIN–ALDOSTERONE SYSTEM IN ADPKD KIDNEYS, INCLUDING ANGIOTENSIIN, ANGIOTENSIN II, RENIN, ACE, AND ANGIOTENSIIN RECEPTORS.\(^\text{15}\) ENHANCED SYMPATHETIC ACTIVITY HAS ALSO BEEN OBSERVED IN ADPKD. A REMARKABLY HIGHER PLASMA CONCENTRATION OF NOREPINEPHRINE AND EPINEPHRINE WAS NOTED IN PATIENTS WITH ESSENTIAL HYPERTENSION, WITH OR WITHOUT RECENT FAILURE.\(^\text{15}\) IN ADDITION, IDIOPATHIC DILATED CARDIOMYOPATHY (IDCM) CHARACTERIZED BY DILATED VENTRICLE AND ATTENUATED SYSTOLIC FUNCTION IS FOUND TO BE ASSOCIATED WITH POLYCYSTIC KIDNEY DISEASE.\(^\text{16}\) IDIOPATHIC DILATED CARDIOMYOPATHY WAS REPORTED TO BE MORE PREVALENT IN HUMAN ADPKD PATIENTS THAN IN THE GENERAL POPULATION, PARTICULARLY IN CASES WITH PKD MUTATIONS, WITH AN APPROXIMATELY 200-FOLD INCREASED PREVALENCE.\(^\text{16}\)

CONSIDERING ACTIVATION OF THE RAAS AND THE SYMPATHETIC SYSTEM, AS WELL AS THE REMARKABLY HIGH INCIDENCE OF LEFT VENTRICULAR HYPERTRPHY, MITRAL VALVULAR PROLAPSE, AND IDCM IN PATIENTS WITH PKD, WE HYPOTHEZISED THAT AF IS CLOSELY ASSOCIATED WITH PKD. DATA REGARDING THE RELATIONSHIP BETWEEN AF AND PKD HAS BEEN LACKING UNTIL NOW. WE CONDUCTED A NATIONWIDE POPULATION-BASED COHORT STUDY TO INVESTIGATE THE RISK OF AF IN PATIENTS WITH PKD.

MATERIALS AND METHODS

DATA SOURCE

WE DESIGNED THIS STUDY AS A POPULATION-BASED RETROSPECTIVE COHORT STUDY BASED ON THE NATIONAL HEALTH INSURANCE RESEARCH DATABASE (NHIRD). THE NHIRD CONTAINS ALL CLAIMS DATA FROM THE TAIWAN NATIONAL HEALTH INSURANCE (NHI) PROGRAM, WHICH IS A SINGLE-PAYER COMPELLARY INSURANCE PROGRAM THAT WAS ESTABLISHED IN 1995. THE TAIWAN NHI COVERED NEARLY 99.9% OF THE 23 MILLION RESIDENTS OF TAIWAN IN 2014. FOR THIS STUDY, WE USED A SUBSET OF THE NHIRD-CONTAINING HEALTH CARE DATA, INCLUDING FILES OF INPATIENT CLAIMS AND THE REGISTRY OF BENEFICIARIES. THE NATIONAL HEALTH RESEARCH INSTITUTES ENCRYPTED THE ORIGINAL IDENTIFICATION INFORMATION AND ASSIGNED ANONYMOUS IDENTIFICATION NUMBERS TO PROTECT PATIENT PRIVACY BEFORE RELEASING THE DATABASE FOR RESEARCH. THIS STUDY WAS APPROVED BY THE ETHICS REVIEW BOARD AT CHINA MEDICAL UNIVERSITY (CMUH104-REC2–115). DISEASE STATUS WAS RECORDED ACCORDING TO INTERNATIONAL CLASSIFICATION OF DISEASES, NINTH REVISION, CLINICAL MODIFICATION (ICD-9-CM) CODES.

STUDY PATIENTS

TO INVESTIGATE THE ASSOCIATION BETWEEN THE RISK OF AF AND PKD, WE CONSTRUCTED A PKD COHORT AND A COMPARISON COHORT (AS SHOWN IN THE SUPPLEMENTAL FIGURE 1, HTTP://LINKS.LWW.COM/MDCM/A656). THE PKD COHORT COMPRISED PATIENTS AGED OLDER THAN 20 YEARS WITH PKD (ICD-9-CM 753.12 AND 753.13), IDENTIFIED FROM INPATIENT CLAIMS FROM 1998 TO 2011. THE INDEX DATE OF THE PKD PATIENTS WAS THE DATE OF THE FIRST DIAGNOSIS FOR PKD. THE COMPARISON COHORT COMPRISED PATIENTS IN THE NHIRD WITHOUT PKD, FREQUENCY MATCHED BY AGE (IN 5-Y BANDS), SEX, AND BASELINE COMORBIDITY OF HYPERTENSION (ICD-9-CM 401–405), CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) (ICD-9-CM 491, 492, AND 496), CONGESTIVE HEART FAILURE (ICD-9-CM 428), DIABETES (ICD-9-CM 250), CHRONIC KIDNEY DISEASE (ICD-9-CM 585), HYPERLIPIDEMIA (ICD-9-CM 272), AND STROKE (ICD-9-CM 430–438) AT A RATIO OF 1:4. THE INDEX DATE OF THE COMPARISON PATIENTS WAS THE SAME YEAR AS THAT OF THE MATCHED CASES, WITH A RANDOMLY ASSIGNED MONTH AND DAY. PATIENTS IN BOTH COHORTS DIAGNOSED WITH AF (ICD-9-CM 427.31) AT THE BASELINE WERE EXCLUDED. OVERALL, 7203 PKD PATIENTS AND 28,739 COMPARISON PATIENTS WERE FOLLOWED-UP UNTIL A DIAGNOSIS OF AF, LOSS TO FOLLOW-UP, DEATH, WITHDRAWAL FROM THE NHI PROGRAM, OR THE END OF 2011.

STATISTICAL ANALYSIS

DISTRIBUTIONS OF AGE (49.4, 50–64, AND ≥65 Y), SEX, AND COMORBIDITIES WERE COMPARED BETWEEN THE PKD AND COMPARISON COHORTS, AND EXAMINED USING THE χ² TEST. THE MEDIAN (INTERQUARTILE RANGE, IQR) AGES AND FOLLOW-UP TIMES OF BOTH COHORTS WERE MEASURED AND COMPARED USING A MANN–WHITNEY U TEST. THE CUMULATIVE INCIDENCE CURVES OF AF WERE ESTIMATED USING THE KAPLAN–MEIER METHOD AND LOG-RANK TEST. THE OVERALL AND SEX-, AGE-, COMORBIDITY-, AND FOLLOW-UP TIME-SPECIFIC INCIDENCE DENSITY RATES (PER 1000 PERSON-Y) WERE CALCULATED FOR BOTH COHORTS. THE IDENTIFICATION OF DEATH EVENTS WAS BASED ON HOSPITAL DISCHARGE BECAUSE OF DEATH AND WITHDRAWAL FROM THE NHI AS INDICATED IN THE NHIRD. AFTER ACCOUNTING FOR THE COMPETING RISKS OF DEATH, THE PKD-TO-COMPARISON-COHORT SUBHazard RATIO (SHR) AND THE 95% CONFIDENCE INTERVALS (CIs) FOR AF WERE ESTIMATED USING UNIVARIABLE AND MULTIVARIABLE COMPETING-RISKS REGRESSION MODELS. THE MULTIVARIABLE MODELS WERE CONCURRENTLY ADJUSTED FOR AGE, AND THE COMORBIDITIES OF HYPERTENSION, COPD, CONGESTIVE HEART FAILURE, DIABETES, CHRONIC KIDNEY DISEASE, HYPERLIPIDEMIA, AND STROKE. FURTHER DATA ANALYSIS WAS CONDUCTED TO EVALUATE THE JOINT EFFECT OF PKD AND AF-ASSOCIATED RISK FACTORS ON THE RISK OF AF. A LOGISTIC REGRESSION ANALYSIS WAS CONDUCTED TO CALCULATE AND COMPARE THE ODDS RATIOS (ORs) OF 30-DAY MORTALITY FROM AF IN THE PKD COHORT COMPARED WITH THE NON-PKD COHORT. DATA MANAGEMENT AND STATISTICAL ANALYSIS WERE PERFORMED USING SAS 9.3 SOFTWARE (SAS INSTITUTE, CARY, NC). THE SIGNIFICANCE LEVEL WAS SET AT A 2-SIDED P VALUE OF LESS THAN .05.

RESULTS

TABLE 1 DISPLAYS THE DEMOGRAPHIC CHARACTERISTICS AND COMORBIDITIES OF THE PKD AND COMPARISON COHORTS. THE MAJORITY OF THE PATIENTS WERE AGED ≥65 YEARS (39.2%) AND MORE THAN HALF WERE WOMEN (APPROXIMATELY 58%). THE MEDIAN AGE WAS 58.7 YEARS IN THE PKD COHORT AND 58.5 YEARS IN THE COMPARISON COHORT. COMORBIDITIES AT THE BASELINE WERE SIMILAR IN THE PKD COHORT THAN IN THE COMPARISON COHORT. THE MEDIAN DURATION OF FOLLOW-UP WAS 3.96 (IQR = 1.53–7.56) YEARS IN THE PKD COHORT AND 4.96 (IQR = 2.21–8.71) YEARS IN THE COMPARISON COHORT. THE KAPLAN–MEIER GRAPH ILLUSTRATES THAT THE CUMULATIVE INCIDENCE OF AF WAS SIGNIFICANTLY HIGHER IN THE PKD COHORT THAN IN THE COMPARISON COHORT (LOG-RANK TEST, P < .001) (SUPPLEMENTAL FIGURE 2, HTTP://LINKS.LWW.COM/MDCM/A656).

IN TOTAL, 247 PKD PATIENTS WERE DIAGNOSED WITH AF, REPRESENTING AN INCIDENCE OF 7.08 PER 1000 PERSON-YEARS, WHEREAS 807 CASES OF AF OCCURRED IN THE COMPARISON COHORT, YIELDING AN INCIDENCE OF 4.98 PER 1000 PERSON-Y, WITH AN ADJUSTED SHR (aSHR) OF 1.31 (95% CI = 1.14–1.51) (TABLE 2). THE OVERALL INCIDENCE AND RISK OF AF WERE COMPARED IN THE PKD COHORT AND THE COMPARISON COHORT REGARDING THE VARIABLES OF SEX, AGE, COMORBIDITY, AND FOLLOW-UP TIME. A RELATIVELY HIGHER RISK OF AF WAS OBSERVED IN THOSE MEN (aSHR = 1.38; 95% CI = 1.16–1.64), THOSE WITH ELDER AGE, BETWEEN 50 AND 64 YEARS (aSHR = 2.33; 95% CI = 1.72–3.17) AND WITHOUT COMORBIDITY (aSHR = 1.46; 95% CI = 1.38–1.51).
### TABLE 1. Characteristics of Patients Between Patients With Polycystic Kidney Disease and Patients Without Polycystic Kidney Disease

| Age, y | Yes (N = 7203) | % | No (N = 28,739) | % | P Value |
|--------|----------------|---|-----------------|---|---------|
| 20–49  | 2312           | 32.1 | 9223           | 32.1 | 0.99    |
| 50–64  | 2065           | 28.7 | 8246           | 28.7 |         |
| ≥65    | 2826           | 39.2 | 11,270         | 39.2 |         |
| Median (IQR) | 58.7 (46.7–72.8) | | 58.5 (45.8–72.2) | | 0.54 |

| Comorbidity | Yes | % | No | % | P Value |
|-------------|-----|---|----|---|---------|
| Hypertension | 2163 | 30.0 | 8606 | 30.0 | 0.89 |
| COPD         | 421  | 5.84 | 1640 | 5.71 | 0.65 |
| Congestive heart failure | 331  | 4.60 | 1279 | 4.45 | 0.60 |
| Diabetes     | 627  | 8.70 | 2486 | 8.65 | 0.88 |
| Chronic kidney disease | 1106 | 15.4 | 4352 | 15.1 | 0.65 |
| Hyperlipidemia | 322  | 4.47 | 1246 | 4.34 | 0.62 |
| Stroke       | 831  | 11.5 | 3288 | 11.4 | 0.82 |

χ² test. COPD = chronic obstructive pulmonary disease. IQR = interquartile range.

### TABLE 2. Incidence of Atrial Fibrillation Stratified by Demographic Characteristics, Comorbidity, and Follow-up Time and Competing Risk (Death) Model Measured Subhazard Ratios for Patients With Polycystic Kidney Disease Compared With Those Without Polycystic Kidney Disease

| Polycystic Kidney Disease | Yes Event PY Rate# | No Event PY Rate# | Crude SHR 1 (95% CI) | Adjusted SHR 1 (95% CI) |
|---------------------------|--------------------|-------------------|-----------------------|-------------------------|
| All                       | 247 34,900 7.08    | 807 161,984 4.98  | 1.27 (1.11, 1.46)***  | 1.31 (1.14, 1.51)***    |
| Sex                       |                    |                   |                       |                         |
| Female                    | 83 15,902 5.22     | 288 71,469 4.03   | 1.14 (0.90, 1.44)     | 1.16 (0.92, 1.48)       |
| Male                      | 164 18,997 8.63    | 519 90,515 5.73   | 1.35 (1.14, 1.60)***  | 1.38 (1.16, 1.64)***    |
| Age, y                    |                    |                   |                       |                         |
| 20–49                     | 18 13,863 1.30     | 40 57,762 0.69    | 1.34 (0.79, 2.27)     | 1.32 (0.77, 2.24)       |
| 50–64                     | 62 10,700 5.79     | 119 49,115 2.42   | 2.31 (1.71, 3.14)***  | 2.33 (1.72, 3.17)***    |
| ≥65                       | 167 10,338 16.2    | 648 55,107 11.8   | 1.36 (1.15, 1.61)***  | 1.36 (1.15, 1.62)***    |
| Comorbidity§              |                    |                   |                       |                         |
| No                        | 101 22,987 4.39    | 260 10,438 2.49   | 1.44 (1.16, 1.80)**   | 1.46 (1.17, 1.82)***    |
| Yes                       | 146 11,913 12.3    | 547 57,636 9.49   | 1.20 (1.00, 1.43)*    | 1.21 (1.01, 1.45)*      |
| Follow time, y            |                    |                   |                       |                         |
| ≤1                        | 66 6453 10.2       | 132 26,901 4.91   | 1.66 (1.25, 2.20)***  | 1.74 (1.31, 2.31)***    |
| >1                        | 181 28,447 6.36    | 675 135,083 5.00  | 1.19 (1.01, 1.39)*    | 1.22 (1.04, 1.43)*      |

CI = confidence interval, PY = person-years.
# Rate = incidence rate per 1000 person-years.
1 Crude SHR, relative subhazard ratio.
2 Adjusted SHR, subhazard ratio adjusted for age, and comorbidities of hypertension, COPD, congestive heart failure, diabetes, chronic kidney disease, hyperlipidemia, and stroke.
3 Comorbidity: patients with any one of the comorbidities (including hypertension, COPD, congestive heart failure, diabetes, chronic kidney disease, hyperlipidemia, and stroke) were classified as the comorbidity group.

P < 0.05.
* P < 0.01.
** P < 0.001.
CI = confidence interval. SHR = subhazard ratio.

### DISCUSSION

In the current study, we observed a significantly higher incidence rate of AF (7.08 per 1000 persons-year) in patients with PKD compared with patients without PKD, which has never been reported until now. After adjustment for the confounding factors of sex, age, and comorbidities, a 1.31-fold increase in the risk of developing AF was observed in patients with PKD.

The cumulative incidence curve with log-rank test showed that patients with more risk factors had a higher rate of new-onset AF. The aSHR of AF in patients with PKD was significantly higher than that of the comparison cohort in the first follow-up year (aSHR = 1.74; 95% CI = 1.31–2.31).

The incidence of AF increased with the number of risk factors. The risk of AF increased from an aSHR of 1.59 (95% CI = 1.15–2.21) to 3.64 (95% CI = 1.93–6.85) when the number of risk factors increased from 1 to more than 5 in comparison with patients without risk factors (Table 3; Figure 1). Mortality from AF in patients with PKD was higher than that in the comparison cohort (Table 4; OR = 1.69; 95% CI = 1.24–2.31).
including hypertension, obesity, diabetes, and hypercholesterolemia, in ADPKD patients. Notably, arrhythmia was the most prevalent self-reported cardiovascular event, occurring in approximately 25.9% of the 419 patients, followed by valvular heart disease; however, the reasons remain to be determined in the previous study.

For patients with PKD, several potential factors have been suggested to be associated with the development of AF, including IDCM, overactivation of the RAAS, autonomic function, and abnormal cardiac handling conditions. The current study is the first to provide clinical evidence of the association between AF and PKD.

Accumulating evidence has shown that cardiac function is directly linked to cardiac-dependent contraction in cardiomyocytes and cardiac dysfunction is closely associated with the mutation of calcium handling proteins. For example, disordered intracellular calcium release channels, such as RyR2, may result in spontaneous leak of calcium from RyR2, consequently leading to arrhythmogenesis. Recently, Anyatonwu et al suggested that mutation of PC2 leads to the calcium alternans associated with RyR2 dysfunction and may be associated with the condition of arrhythmia. Polycystin-2 has been regarded as an intracellular calcium channel that inhibits RyR2 expression, alters intracellular Ca2+ signaling, and is a key contributor to AF-maintaining substrates.

In addition, previous experimental data have shown that the defects of PC2 may be associated with altered calcium signaling, desensitized calcium-contracture coupling in cardiomyocytes, and abnormal changes in intracellular calcium. Furthermore, attenuated sensitivity to calcium in myofibrils has been suggested to be associated with the subtle diastolic dysfunction and IDCM in patients with PKD, which has been considered to be independent of the effect of hypertension and chronic renal failure. Young normotensive ADPKD patients have been found to be associated with the development of biventricular diastolic dysfunction, which may further support the assumption. In addition, PC2 deficiency has been suggested to be associated with changes in the beta-adrenergic signaling pathway. Therefore, defects in polycystin are closely associated with the remodeling of the heart in PKD patients, particularly in the absence of renal failure and high blood pressure.

Collectively, our findings suggest that the abnormal calcium handling caused by the deficiency of PC2 is associated with the risk of AF in patients with PKD.

We also determined other risk factors for AF in the PKD cohort. We observed that congestive heart failure, hypertension, and age were independent risk factors associated with the development of AF in the patients with PKD. Furthermore, a graded trend of AF risk was noted in the PKD patients when the number of risk factors increased in the PKD patients. The incidence rate of AF was 4.39 per 1000 person-years for the PKD patients without comorbidities and significantly increased to 39.6 per 1000 person-years for patients with 5 or more comorbidities, who exhibited an approximately 3.64-fold increase in AF prevalence compared with those without any comorbidity. Previous studies have shown that several crucial risk factors are associated with new-onset AF, including advanced age, hypertension, and congestive heart failure. Most of these factors can result in the electrical and structural remodeling of the heart chamber and may contribute to the development and maintenance of AF. In our study, these factors were more prevalent in the patients with PKD; therefore, the risk stratification of AF incidence can assist clinicians in identifying patients at a high risk of developing AF. The congenital defects of patients with PKD and the most prevalent risk factors for AF may account for the findings of the current study.

In addition, a 1.69-fold increased risk of mortality was significantly associated with AF in the PKD cohort. Previous studies have revealed that AF may cause a variety of complications and mortality in the general population and our findings showed a consistent effect of AF on mortality in PKD patients.

The strengths of our study are its population-based design, generalizability of findings, and use of population-based data and NHIRD records using a large sample size and having low loss to follow-up in the longitudinal design, including study and control cohorts. In addition, NHIRD covers a highly representative sample of Taiwan’s general population because the reimbursement policy is universal and operated by a single buyer, the government in Taiwan. All insurance claims should be scrutinized by medical reimbursement specialists and peer review according to the standard diagnosed criteria in the study. Therefore, the diagnoses of PKD based on ICD-9 codes in this study were highly reliable.

Some limitations of the current study should be clarified. First, data regarding some risk factors, such as tobacco smoking, alcohol consumption, and physical activity cannot be obtained from the NHIRD, as mentioned previously. Second, biologic data, including echocardiographic parameters, and some crucial data on preexisting conditions such as thyroid dysfunction were also lacking. To overcome these limitations, associated factors such as COPD, coronary artery disease, and stroke were included in the multivariable Cox proportional hazard models. Third, although the diagnostic accuracy of the NHIRD has been validated in previous studies, 24-hour Holter monitoring is not routinely used for all AF diagnoses. Hence, asymptomatic AF is highly likely to have been underestimated in the study. Finally, data regarding the genetic analysis of PC1 and PC2 are lacking in the database, although this does not hinder the diagnosis of PKD.

In conclusion, AF is a common sustained arrhythmia in daily practice and often leads to multiple comorbidities and poor prognosis, which eventually increase health care costs and mortality. Based on the findings of current study, we suggest that AF is associated with PKD patients. Notably, a remarkably high incidence rate and risk were observed in patients with PKD when multiple risk factors were combined. A high index of suspicion should be maintained when examining PKD patients with irregular betas. Early prophylactic therapy is warranted in these patients.

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