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НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

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ASSOCIATION OF CHRONIC KIDNEY DISEASE WITH ATRIAL FIBRILLATION

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✓ ***Resume***

Chronic kidney disease (CKD) is closely associated with an increased risk of atrial fibrillation (AF). Renal dysfunction may contribute to the development and progression of AF through inflammation, oxidative stress, electrolyte imbalance, and cardiovascular remodeling. Early identification of AF risk in patients with CKD may improve cardiovascular outcomes and enable timely preventive strategies.

Keywords: chronic kidney disease, atrial fibrillation, renal dysfunction, cardiovascular risk, inflammation, arrhythmia.

ВЗАИМОСВЯЗЬ ХРОНИЧЕСКОЙ БОЛЕЗНИ ПОЧЕК С ФИБРИЛЛЯЦИЕЙ ПРЕДСЕРДИЙ

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✓ ***Резюме***

Хроническая болезнь почек (ХБП) тесно связана с повышенным риском развития фибрилляции предсердий (ФП). Нарушение функции почек может способствовать возникновению и прогрессированию ФП посредством воспаления, окислительного стресса, электролитных нарушений и ремоделирования сердечно-сосудистой системы. Раннее выявление риска ФП у пациентов с ХБП может способствовать улучшению сердечно-сосудистых исходов и своевременному проведению профилактических мероприятий.

Ключевые слова: хроническая болезнь почек, фибрилляция предсердий, почечная дисфункция, сердечно-сосудистый риск, воспаление, аритмия.

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✓ ***Rezyume***

Surunkali buyrak kasalligi (SBK) bo'lmachalar fibrillyatsiyasi (BF) rivojlanish xavfining oshishi bilan chambarchas bog'liq. Buyrak faoliyatining buzilishi yallig'lanish, oksidlovchi stress, elektrolitlar muvozanatining buzilishi va yurak-qon tomir tizimining qayta tuzilishi orqali BF rivojlanishi va kuchayishiga olib kelishi mumkin. SBK bilan og'rigan bemorlarda BF xavfini erta aniqlash yurak-qon tomir asoratlarini kamaytirish va profilaktik choralarni o'z vaqtida amalga oshirishga yordam beradi.

Kalit so'zlar: surunkali buyrak kasalligi, bo'lmachalar fibrillyatsiyasi, buyrak disfunksiyasi, yurak-qon tomir xavfi, yallig'lanish, aritmiya.

Relevance

The incidences of atrial fibrillation (AF) and chronic kidney disease (CKD) are increasing, and AF is prevalent in patients with CKD. However, few studies have investigated the incidence or association of AF in a large CKD population from a longitudinal study.

METHODS AND RESULTS: During a median follow-up of 2.1 years, the annual incidence rate of AF was 1.17 per 1000 person-years among subjects without CKD, 1.55 for stage 1 CKD, 1.86 for stage 2 CKD, 2.1 for stage 3 CKD, and 4.33 for stage 4 CKD. In Fine-Gray regression models, CKD was associated with an increased risk of AF; the adjusted hazard ratios and 95% CIs of AF occurrence were 1.77 (1.69–1.85), 1.85 (1.80–1.91), 1.99 (1.95–2.04), and 4.04 (3.07–5.33) in individuals with CKD stages 1, 2, 3, and 4, respectively, compared with non-CKD. The association between CKD and incident AF remained statistically significant after adjustment for multiple confounding factors and was consistent across subgroups stratified by sex and age.

CKD is associated with an increased incidence of AF. Even mild CKD is associated with incident AF, and there was a stepwise increase in the risk of incident AF with a decrease in renal function.

Atrial fibrillation (AF) is one of the most common forms of cardiac arrhythmia[1] and is associated with cerebrovascular and cardiovascular complications resulting in significant morbidity and mortality.[2–5] Furthermore, increased prevalence and incidence of AF represent a significant global health care economic burden.[6]

Chronic kidney disease (CKD) is defined as ab-normality of kidney structure or renal function lasting for >3 months with implications on health.[7] CKD is a rapidly growing public health problem, and a meta-analysis reported an estimated global CKD prevalence of 13.4%.[8,9] Patients with CKD exhibit an elevated car-diovascular risk manifesting as not only heart failure, coronary heart disease, and mortality, but also AF.[10–12] AF and CKD are closely connected conditions that share common risk factors such as hypertension, diabetes, and coronary artery disease.[13,14] CKD activates the renin–angiotensin–aldosterone system and sympathetic nervous system, which induce oxidative stress, systemic inflammation, and volume overload.[15] Angiotensin II can increase atrial pressure, accelerate atrial fibrosis, and cause ion channel dysfunction; all of which are related to structural and electrical remodeling of the atria, thus resulting in AF.[16,17] Moreover, poor blood pressure control is both a cause and ef-fect of CKD and affects the vast majority of patients with CKD.[18] Left ventricular hypertrophy is secondary to both pressure and volume overload in patients with CKD, which contributes to diastolic dysfunction and is another independent risk factor for incident AF.[19,20] These factors are highly correlated with atrial electrical and structural remodeling, contributing to the development of AF. The growing prevalence of both AF and

CKD suggests that more patients will suffer from con-current conditions. Therefore, we have to understand the importance of the implications of coexisting AF and CKD.

The aim of the study was to examine the relationship between chronic kidney disease and atrium fibrillation.

Materials and methods

As mentioned, CKD has been demonstrated to be an independent risk factor contributing to the higher incidence and prevalence of AF, but there were important limitations to the preceding studies involving patients with AF in CKD. The small number of recruited subjects resulted in insufficient power to discriminate differences of AF incidence in CKD compared with non-CKD. Moreover, to date, many of studies have been targeted to Western populations. Therefore, the aim of the current study is to evaluate the development of AF across CKD stages, compared with subjects without CKD, in a nationwide Asin population representative study with longitudinal follow-up. To do so, we analyzed data from participants enrolled in the National Health Insurance Database (NHID).

We screened adult (≥40 years of age) health examinees who received 2 or more checkups from 2009 to 2012, and extracted 70% of subjects by random sampling due to the limit of data capacity set by the NHIS. According to Kidney Disease Improving Global Outcomes guidelines, CKD is classified according to the extent of estimated glomerular filtration rate (eGFR) and albuminuria.[30] The presence of incident CKD was defined by the presence of dipstick albumi-nuria ≥ +1 or eGFR <60 mL/min per 1.73 m2, calcu-lated from the Chronic Kidney Disease Epidemiology Collaboration creatinine equation.[31] CKD stages were defined using the recommendations of the National Kidney Foundation.[32]

CKD stage 1 (eGFR ≥90 mL/min per 1.73 m2) and stage 2 (eGFR ≥60 and <90 mL/min per 1.73 m2) included subjects who had dipstick albu-minuria ≥+1 but had normal or mildly reduced eGFR at baseline. Subjects with CKD stage 3 (eGFR ≥30 and <60 mL/min per 1.73 m2) and stage 4 (eGFR ≥15 and <30 mL/min per 1.73 m2) were also identified.

Subjects who met the CKD criteria at least once among several health checkups were classified as CKD. The initial health checkup date that met the definition of CKD was considered as the baseline date. We also defined non-CKD subjects as those who did not meet CKD criteria in every health checkup, and their first checkup date was designated as the baseline date. We inserted the washout period of 1 year from baseline date to exclude the subjects who had any out-come events within the 1-year period after the baseline date. We defined the index date as the date of starting follow-up, which means 1 year after the baseline date.

Subjects were excluded if baseline values of eGFR or dipstick albuminuria were missing or eGFR at baseline date was an outlying value (defined as <15 mL/min per 1.73 m2 or >99.9th percentile) to minimize labora-tory error. To exclude patients with valvular AF, those who were diagnosed with mitral stenosis or had pre-existing mechanical heart valves before the baseline date were also excluded. Because we intended to confirm de novo AF in patients with CKD, those who were already diagnosed with AF before the baseline date were excluded. We inserted the washout period of 1 year after baseline date to analyze the association of CKD more accurately to AF occurrence by excluding those who were diagnosed with AF, end-stage kidney disease, or died of any cause during the washout period. The comorbid diseases were identified by ICD-10 diagnostic codes, special certification codes for each disease, and NHIS claims data (Data S1). Therefore, the final study population comprised 4 827987 partic-ipants (Figure S1).

Data Collection and Measurements Demographic and clinical characteristics on the bassline date were collected as baseline variables. Baseline information, including age, sex, body mass index, sys-tolic/diastolic blood pressure, history of smoking and drinking, comorbidities, and laboratory findings, were collected. We stratified alcohol consumption into these 6 subgroups based on a self-reported questionnaire: unknown, nondrinker, occasional moderate (<6 drinks per week and <5 drinks on occasion), regular moder-ate (≥6 drinks per week and <5 drinks on occasion), occasional binge (<6 drinks per week and ≥5 drinks on occasion), and regular binge (≥6 drinks per week and ≥5 drinks on occasion). The underlying comorbidi-ties of hypertension, diabetes, and dyslipidemia were defined as follows. Hypertension was defined as the combination of a previous diagnosis of hypertension, corresponding ICD-10 diagnostic codes (I10–I15) within 1 year before the index date, self-reported previous di-agnosis of hypertension, or measured blood pressure ≥140/90 mm Hg at the health checkup. Diabetes was defined as the combination of a previous diagnosis of diabetes, corresponding ICD-10 diagnostic codes (E10–E14) within 1 year before the index date, self-reported diagnosis of diabetes, history of prescribing diabetes medication or insulin within 1 year before the index date, or fasting glucose ≥126 mg/dL at the health checkup. Dyslipidemia was defined as the combina-tion of a previous diagnosis of dyslipidemia with corre-sponding ICD-10 diagnostic code (E78), self-reported diagnosis of dyslipidemia, or serum total cholesterol ≥240 mg/dL. The definitions of these comorbidities were.

To compare the differences among the CKD groups, continuous variables were summarized with means and SDs and analyzed using the ANOVA test. Categorical variables were presented as frequencies and percentages, and the $\chi 2$ test or Fisher exact test was used to compare the values. Incidence rates of AF were expressed as events per 1000 person-years. We account for the competing risk of death. The cumulative incidence function with the gray test and Fine-Gray regression models was performed to examine the association between development of atrial fibrillation during follow-up. The results are provided as subhaz-ard ratios with 95% CI. Model 1 included adjustment for age, sex, body mass index, smoking, and drinking. Model 2 included further adjustment for systolic blood pressure, diastolic blood pressure, and comorbidities. Several predisposing factors for AF have been identified. There has been an accumulation of evidence on sex-specific and age-specific differences in incidence, prevalence, and outcomes of patients with AF. With this rationale, we analyzed incident AF with sub-group analysis with sex (men versus women) and age (<65 versus ≥65 years of age) to determine whether CKD can affect incident AF even after considering the predisposing factors of AF. Statistical analyses were performed using SAS version 9.2 (SAS Institute, Cary,NC).

Results and discussions

The main age of the study population was 51.9±7.8 years, and 50.6% were men. Subjects with more advanced CKD stages were older and more likely to have anemia and other comorbid diseases such as diabetes and hypertension.

The Figure shows Fine-Gray cumulative incidence function curves of incident AF for up to 8 years ac-cording to the CKD stage. Higher stages of CKD were associated with a higher risk of AF development. The incidence rates of AF were 1.17 per 1000 person-years for subjects without CKD, 1.55 for CKD stage 1, 1.86 for stage 2, 2.10 for stage 3, and 4.33 for stage 4 (Table 2). CKD was associated with an increased risk of AF; adjusted hazard ratios (HRs) and 95% CIs for de novo AF were 1.77 (1.69–1.85), 1.85 (1.80–1.91), 1.99 (1.95–2.04), and 4.04 (3.07–5.33) in individuals with CKD stages 1, 2, 3, and 4, respectively, after adjustment in Model 2 (Table 2). There was no significant effect modification by sex or age when considering a more advanced stage of CKD had a trend with a higher risk of AF (P values for interaction: P=0.672 and P=0.646, respectively). After stratifying the subjects according to sex, we found the incidence rate was increased across CKD stage in both sexes. More advanced stages of CKD had a trend with a higher risk of AF development in both sex groups. The risk of incident AF in subjects with stage 4 was approximately 4 times that of sub-jects without CKD in both sexes (HR, 4.24 [95% CI, 3.34–5.37]; P<0.001 for men; HR, 3.98 [95% CI, 2.14–7.40]; P<0.001 for women) after adjusting for multiple variables.

We stratified the subjects based on their age as of the index date (age <65 or ≥65 years of age). AF inci-dence rate increased across CKD stages in both age groups (Table 4). More advanced stages of CKD had a trend with a higher risk of AF development in both age groups. The risk of AF development in CKD stage 4 was approximately 3 times higher in subjects with-out CKD in the <65 years age group (HR, 3.27 [95% CI, 2.24–4.76]; P<0.001) and 2 times higher in the≥65 years age group (HR, 3.86 [95% CI, 3.03–4.92]; P<0.001) after adjusting for multiple variables.

The incidence of AF was highest in stage 4, and the association between CKD stage and AF persisted after multivariable adjustment and was consistent throughout subgroup analysis stratified by age and sex.

The prevalence and incidence of AF were high among patients with CKD and increased with higher CKD stages, which was supported by previous results. Although further research is needed to investigate the biological mechanism explaining the re-lationship between CKD and AF, we found a strong as-sociation between CKD and incident AF in this study. The association between renal dysfunction and risk of AF had been explored in several prior studies, although not all studies found an association. In a recent study,

AF showed a causal effect on kidney function, but the study found that kidney function was difficult to use to predict the occurrence of AF. This may be due to the limited number of subjects who had severe renal dysfunction. Moreover, the causal effect analyzed by genetic instrument had limitations in the analytic method itself, so there still remained a possibility of effect of renal function on AF. In the PREVEND (Prevention of Renal and Vascular End-stage Disease) study, no association was shown between incidence of AF and markers of renal function such as creatinine, eGFR, and cystatin C. The PREVEND study oversampled the subjects with albuminuria to investigate the cardiovascular and renal outcomes of albuminuria subjects, whereas the present study was based on the general population, showing the association of CKD and AF. In the CHS (Cardiovascular Health Study), no relation between incidence or prevalence of AF and eGFR was observed. However, there was a limitation to generalizing the results to all ages, because the study was only targeted at the elderly, with an average age of ≈75 years. Our analysis differs from the previous studies. Second, we excluded subjects previously diagnosed with AF and followed the study subjects for up to 8 years to investigate the development of de novo AF. To date, many of the studies have been cross-sectional studies tak-ing a snapshot of a temporal relationship, from which it is hard to establish an association between AF and CKD. With long-term follow-up duration, we found that there was an incremental increase of incidence and risk of AF according to CKD stages, which was difficult to find in a short-term follow-up period.

Most of the epidemiological reports on AF from CKD have been derived from Western populations. Therefore, the risk of AF among groups of different ethnicities should be evaluated with caution, because studies on the clinical epidemiology of AF in non-Western groups are limited. Previous studies presenting global and regional differences in the prevalence of AF reported a lower prevalence of AF

among the Asian population compared with the Western population. The different incidences of AF between Asian and Western patients with CKD might be attributed to different genetic backgrounds, diet, and lifestyles between Asian and Western coun-tries. According to the ARIC (Atherosclerosis Risk in Communities) cohort study, which targets the US population, the incidence rates of AF were 10.2 per 1000 person-years in CKD stage 3 and 15.2 per 1000 person-years in CKD stage 4.

Based on a Japanese population study, the incidence rate of AF, confirmed by ECG, was 2.2 per 1000 person-years at CKD stages 1 and 2, 5.1 at CKD stage 3, and 6.6 at CKD stages 4 and 5. Possible explanations for the discrepancy between the Japanese study and ours include the older age of the subjects in the Japanese study (average age of 63 years versus 52 years in our study) and the method used to define AF.

In addition to evaluating the overall incidence of AF in CKD, we analyzed incident AF with subgroup analysis. The association between the severity of CKD and incident AF showed a robust relationship across several subgroup analyses and after multivariable adjustments. The incidence of AF in the general population has been estimated to be 1.5- to 2.0-fold higher in men than in women. Therefore, we analyzed the difference in incidence of AF across the stages of CKD after stratification by sex. We showed that the incidence rate of AF increased across CKD stages in both sexes. We also analyzed incident AF after stratification by age. Aging is the most important risk factor of AF. We stratified the study subjects into younger (<65 years of age) and older (≥65 years of age) groups, because there is a marked increase in AF between 60 and 65 years of age. Due to the characteristics of the national health checkup population, our study subjects were predominantly <65 years of age (n=4452329, 92.1%). We showed that the incidence rate of AF was increased across CKD stages in both age groups. The association between CKD stage and AF was consistent across both sex and age subgroups.

Our study had several limitations. First, this study was an observational study, and there were inherent limitations such as hidden confounding factors. Second, this study was a nationwide, population-based retrospective observational study, which was susceptible to several biases including selection bias. The NHIS provides biennial health checkups to all health insurance subscribers, and the study subjects most likely in-cluded those who maintain healthier lifestyles or those who are more concerned about their health. In other words, patients with advanced CKD tend to have regular hospital visits rather than regular health checkups; thus, such patients might be excluded from this study, also resulting in selection bias. Third, the incidence of AF was based entirely on claims data, and there could have been undetected or unreported episodes of AF. Population studies using claims data inherently do not directly screen the ECG, so there is a possibility of underestimation of silent AF. Conversely, misclassification of AF diagnostic codes could lead to overes-timation of AF. However, the method of identifying AF using ICD-10 diagnostic codes in systemic studies has been statistically verified, and others have previously shown that the validity of AF ascertainment using hospitalizations is acceptable. Fourth, measurement of the baseline serum creatinine and urine dipstick pro-tein may not be representative of kidney function. We defined the subjects who met the CKD criteria at least a single time from a regular health checkup. A few sub-jects with temporarily reduced renal function or albuminuria might have been classified to CKD. It could be hard to reflect on the chronicity of CKD. This may lead to random measurement error and regression dilution bias, which tends to underestimate the real associa-tion between CKD and AF development. However, it is an exceptional situation that acutely ill patients with acute kidney injury receive regular health checkups. Changes in the level of renal function or albuminuria during the follow-up may also dilute the impact of CKD on the risk of AF development, which tends to under-estimate the real association between CKD and AF de-velopment. We could say the incidence or hazard ratio of AF to CKD would be more intense in the real world. Fifth, according to our study design, there would be some concern about occurrence of immortal time bias to CKD, because the first measurement of eGFR or albuminuria defined the CKD. However, as the length of follow-up and the total population size increases, the proportion of the group, which was misclassified to CKD, becomes a smaller and smaller portion of the total person-time denominator, and the magnitude of immortal time bias diminishes. Despite these several limitations, this study is meaningful in that this is the first big data study on a nationwide scale to follow sub-jects for 10 years and observe the incidence of de novo AF in patients with CKD.

Conclusion

In conclusion, CKD was associated with an in-creased incidence of AF in this large population-based study. This association was present among patients with CKD stages from 1 to 4, remained consistent across several subgroups, and persisted after adjustment for multiple potential confounders. Patients with CKD need more intensive monitoring for AF to prevent subsequent AF complications.

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