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Stage-Dependent Atrial Fibrillation Risk in Gastric Cancer: A Nationwide SEER-Based Cohort Study in South Korea

Jin-Ho Park[1]*, So-Yeon Kim[1], Min-Su Choi[1]

[1]Department of Oncology, College of Medicine, Seoul National University, Seoul, South Korea.

***E-mail** ✉ jh.park.research@gmail.com

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ABSTRACT

Compared with the general population, individuals with gastric cancer (GC) experience higher rates of atrial fibrillation (AF) and cardiovascular death. Yet, the influence of cancer stage on AF development has not been well defined. This research assessed how GC stage, classified by the Surveillance, Epidemiology, and End Results (SEER) system, relates to AF risk. A retrospective cohort analysis was conducted using anonymized data from South Korea's Cancer Public Library Database, including patients diagnosed with GC between 2012 and 2019 and followed until 2020. The likelihood of AF occurrence was analyzed across SEER categories (localized, regional, distant) by estimating adjusted hazard ratios (aHRs) with 95% confidence intervals (CIs). Stratified analyses were performed according to age, gender, diagnosis year, and comorbid diseases. Among 211, 500 eligible participants, 7266 developed AF during follow-up. A stage-dependent trend was observed, where AF risk rose with disease progression: aHR 2.00 (95% CI 1.81-2.22) in distant-stage and 1.32 (95% CI 1.25-1.41) in regional-stage GC, compared with localized cancer. These trends were consistent across all subgroups and particularly marked among younger patients, females, and those without hypertension. AF incidence in GC patients correlates with the initial cancer stage. Intensive AF screening and preventive management may be beneficial for individuals with advanced-stage GC to improve long-term outcomes.

Keywords: Gastric carcinoma, Atrial fibrillation, SEER classification, Epidemiologic risk, Cancer stage

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Introduction

Gastric cancer (GC) remains a major global health concern, ranking fifth in both incidence and mortality worldwide [1]. In South Korea, GC represents the most common malignancy and the fourth most frequently diagnosed cancer, with an incidence of 57.2 per 100, 000 in 2021 [2]. Improvements in therapeutic strategies have led to extended survival, shifting clinical focus toward managing long-term complications and comorbidities among GC survivors [3].

Recent epidemiologic findings suggest that GC patients face a higher probability of developing cardiovascular diseases such as atrial fibrillation (AF) and exhibit greater cardiovascular mortality than the general population [4, 5]. The link between AF and malignancies has been documented across various tumor types [6-11]. Shared determinants—such as aging, obesity, tobacco use, and male gender—as well as biological pathways involving systemic inflammation and neurohormonal activation, likely contribute to this relationship. Additionally, cancer therapies, including chemotherapy and radiation, may further predispose patients to AF onset [10, 11].

Despite these insights, evidence is limited regarding how AF risk differs according to GC stage at diagnosis. Since both cancer-related inflammation and treatment toxicity intensify with disease advancement, we posited that individuals presenting with later-stage GC would face a greater likelihood of AF. Accordingly, this investigation explored AF incidence across GC stages at the time of diagnosis.

Materials and Methods

Study data and population

Data were drawn from the Cancer Public Library Database of South Korea, encompassing newly diagnosed cancer cases from 2012 to 2019 [12]. GC patients were identified using ICD-O-3 code C16, yielding an initial sample of 235, 167 cases. Exclusion criteria were: age <30 years (N = 673), missing SEER stage information (N = 11, 769), incomplete covariates (N = 4234), and pre-existing AF (ICD-10 I48) prior to GC diagnosis (N = 6991). After exclusions, 211, 500 patients remained for analysis **(Figure 1)**.

The index date corresponded to each patient's GC diagnosis. Follow-up continued until AF onset, death, or 31 December 2020, ensuring at least one year of observation for all subjects.

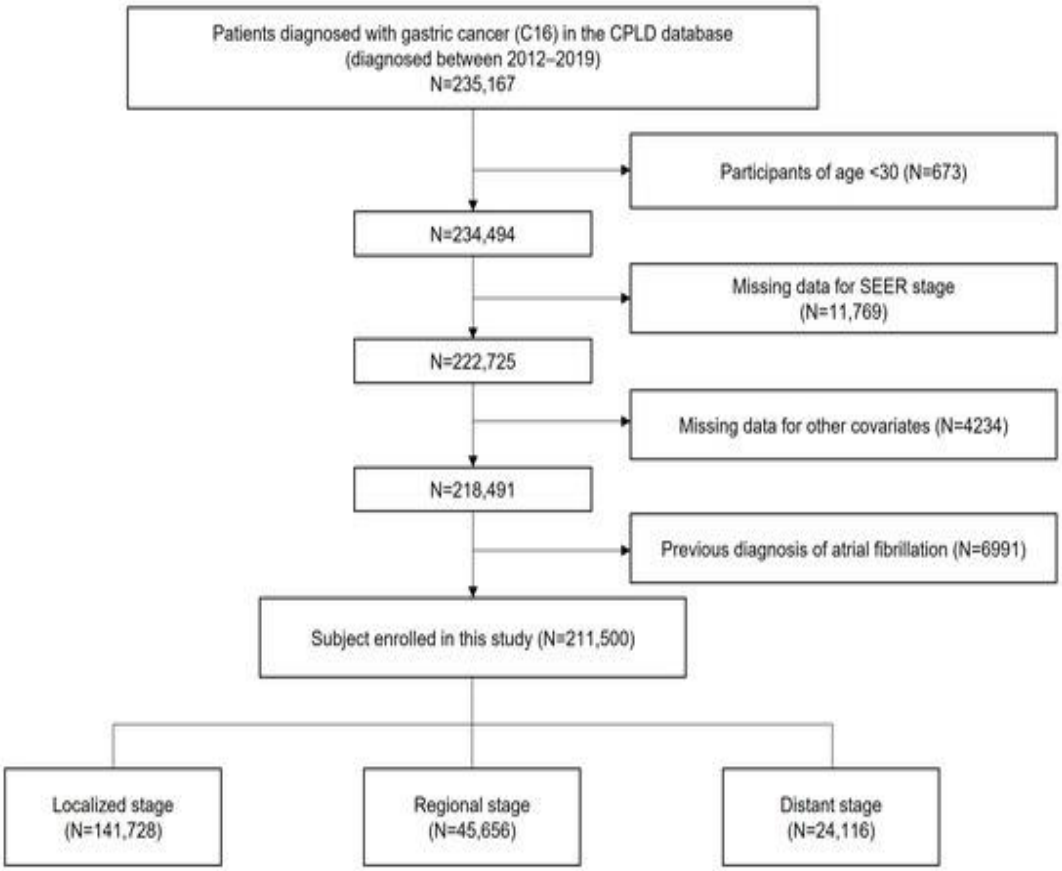

Figure 1. Flow diagram of the inclusion and exclusion process.

Ethical approval was waived by the Institutional Review Board of Seoul National University (IRB No. 2411-121-1590), given that the study used de-identified retrospective data and required no patient consent.

Operational definitions

The classification of gastric cancer (GC) followed the Surveillance, Epidemiology, and End Results (SEER) system, dividing cases into localized, regional, and distant categories.

Information on initial management—including surgery, radiotherapy, chemotherapy, and immunotherapy—administered within four months after diagnosis was obtained.

The main endpoint was the occurrence of atrial fibrillation (AF), identified using ICD-10 code I48, confirmed either during a hospital admission or at two or more outpatient visits.

Statistical approach

Continuous variables were summarized as means with standard deviations and evaluated using ANOVA, while categorical data were displayed as percentages and analyzed using the χ^2 test.

The risk of AF across SEER stages of GC was examined through five Cox proportional hazards models:

Model 1: Unadjusted.

Model 2: Adjusted for sex and age.

Model 3: Model 2 + income and region.
Model 4: Model 3 + hypertension, diabetes, and dyslipidemia.
Model 5: Model 4 + initial therapy.
Kaplan-Meier survival curves were generated to visualize the cumulative AF incidence, and log-rank tests were applied to compare differences among stages.
Subgroup analyses were stratified by age, sex, diagnosis period, and comorbidity profile.
A two-sided $p < 0.05$ denoted statistical significance.
All analyses were performed using SAS 9.4 (SAS Institute, Cary, NC, USA) and R 4.4.1 for Windows.

Results and Discussion

Study population and baseline data
Baseline patient characteristics across SEER stages are presented in **Table 1**.
Among 211, 500 participants, 141, 728 had localized, 45, 656 had regional, and 24, 116 had distant disease.
Those with localized GC tended to be younger and more frequently male, with the majority aged 40-64 years.
Surgical intervention was the most common initial therapy in the localized and regional groups, whereas chemotherapy predominated among patients with distant-stage cancer.
Hypertension and dyslipidemia were more prevalent in the localized group compared with the advanced stages.
The mean observation period for all patients was 3.89 ± 2.58 years, with progressively shorter follow-up durations in higher-stage disease **(Table 1)**.

Table 1. Baseline data for patients categorized by SEER stage.

	No. (%)				
	Total Population (N = 211, 500)	**SEER Summary Stage**			***p*-Value**
		Localized (N = 141, 728)	**Regional (N = 45, 656)**	**Distant (N = 24, 116)**	
Age at diagnosis (years)					<0.0001
30-39	5682 (2.69)	3171 (2.24)	1437 (3.15)	1074 (4.45)	
40-64	103, 642 (49)	71, 489 (50.44)	20, 968 (45.93)	11, 185 (46.38)	
≥65	102, 176 (48.31)	67, 068 (47.32)	23, 251 (50.93)	11, 857 (49.17)	
Sex					0.0002
Male	143, 244 (67.73)	96, 397 (68.02)	30, 606 (67.04)	16, 241 (67.35)	
Female	68, 256 (32.27)	45, 331 (31.98)	15, 050 (32.96)	7875 (32.65)	
Initial treatment					
Surgery	174, 298 (82.41)	129, 859 (91.63)	38, 753 (84.88)	5686 (23.58)	<0.0001
Chemotherapy	42, 532 (20.11)	6972 (4.92)	20, 401 (44.68)	15, 159 (62.86)	<0.0001
Radiotherapy	1628 (0.77)	180 (0.13)	606 (1.33)	842 (3.49)	<0.0001
Immunotherapy or hormone therapy	214 (0.1)	69 (0.05)	43 (0.09)	102 (0.42)	<0.0001
Year of diagnosis (year)					
2012-2013	53, 749 (25.41)	34, 888 (24.62)	12, 598 (27.59)	6263 (25.97)	
2014-2015	52, 526 (24.83)	34, 878 (24.61)	11, 576 (25.35)	6072 (25.18)	
2016-2017	53, 845 (25.46)	36, 683 (25.88)	11, 115 (24.35)	6047 (25.07)	
2018-2019	51, 380 (24.29)	35, 279 (24.89)	10, 367 (22.71)	5734 (23.78)	
Comorbidities					

Diabetes mellitus	43, 637 (20.63)	28, 880 (20.38)	9707 (21.26)	5050 (20.94)	<0.0001
Hypertension	94, 894 (44.87)	63, 970 (45.14)	20, 540 (44.99)	10, 384 (43.06)	<0.0001
Dyslipidemia	57, 169 (27.03)	41, 558 (29.32)	10, 787 (23.63)	4824 (20)	<0.0001
Income level					<0.0001
Medical aid + 1st and 2nd decile	41, 181 (19.47)	26, 206 (18.49)	9671 (21.18)	5304 (21.99)	
3rd to 8th decile	105, 975 (50.11)	70, 187 (49.52)	23, 400 (51.25)	12, 388 (51.37)	
9th and 10th decile	64, 344 (30.42)	45, 335 (31.99)	12, 585 (27.56)	6424 (26.64)	
Residential area, urban	91, 103 (43.07)	61, 443 (43.35)	19, 330 (42.34)	10, 330 (42.83)	0.0005
Follow-up duration (years)	3.89 ± 2.58	4.49 ± 2.41	3.47 ± 2.55	1.12 ± 1.37	<0.0001

SEER—Surveillance, Epidemiology, and End Results; BMI—body mass index. Continuous values shown as mean ± SD; categorical as n (%).

Association between GC Stage and AF

During follow-up, 4765 localized, 1754 regional, and 747 distant-stage cases of AF were identified, corresponding to incidence rates of 7.47, 11.06, and 27.73 per 1000 person-years, respectively **(Table 2)**.

Table 2. Incidence and hazard ratios of AF by SEER stage.

SEER Stage	**N**	**Event**	**IR [a]**	**HR (95% CI)**				
				Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Localized	141, 728	4765	7.49	1 (Ref.)	1 (Ref.)	1 (Ref.)	1 (Ref.)	1 (Ref.)
Regional	45, 656	1754	11.06	1.39 (1.32-1.47)	1.38 (1.30-1.45)	1.38 (1.30-1.45)	1.39 (1.32-1.47)	1.32 (1.25-1.41)
Distant	24, 116	747	27.73	2.29 (2.11-2.48)	2.33 (2.15-2.52)	2.33 (2.15-2.52)	2.39 (2.20-2.59)	2.00 (1.81-2.22)
***p*-value**								

SEER: Surveillance, Epidemiology, and End Results; IR: incidence rate; HR: hazard ratio; CI: confidence interval.
Model 1: Unadjusted; Model 2: Adjusted for age and sex; Model 3: + income, residence; Model 4: + comorbidities; Model 5: + initial treatment.
a Number of cases per 1000 person-years.
Across all statistical models, a progressive elevation in AF risk was seen with advancing GC stage.

In the fully adjusted Model 5, which accounted for demographic, socioeconomic, clinical, and treatment factors, the aHR reached 1.32 (95% CI 1.25-1.41) for the regional group and 2.00 (95% CI 1.81-2.22) for the distant group, compared to the localized reference.

The Kaplan-Meier plots showed a clear gradient, with higher AF incidence at more advanced GC stages **(Figure 2)**.

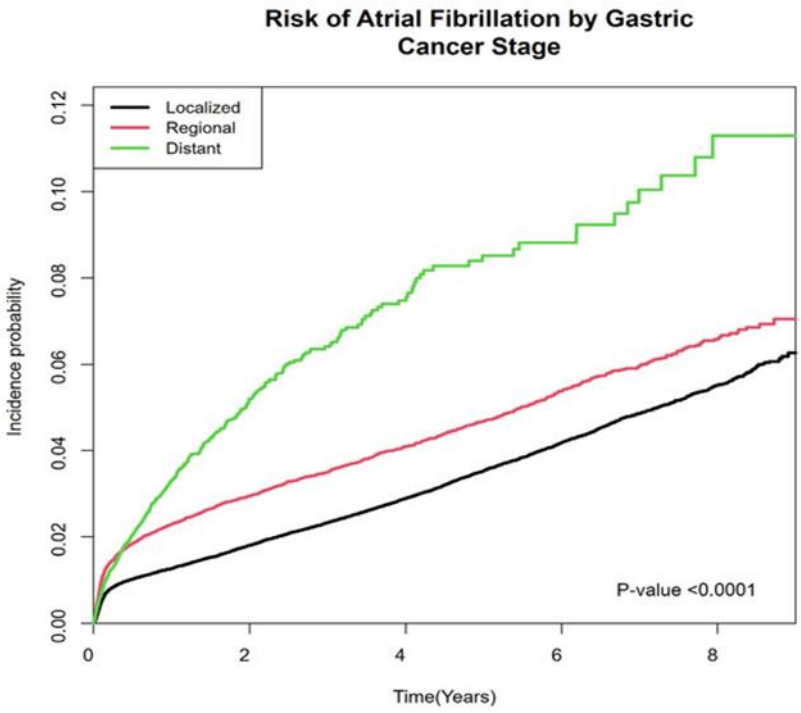

Figure 2. Kaplan-Meier curves showing cumulative AF incidence by SEER-defined GC stage.
SEER: Surveillance, Epidemiology, and End Results.

Subgroup evaluation

The subgroup analyses confirmed that patients diagnosed at later stages consistently showed greater AF risk regardless of sex, age, comorbidity status, or year of diagnosis **(Table 3)**.

The association was especially strong among younger patients, females, and individuals without hypertension. Furthermore, those diagnosed between 2016 and 2019 exhibited a tighter stage-AF association than patients diagnosed from 2012 to 2015.

Table 3. Subgroup analysis of AF incidence across SEER stages.

		SEER Stage	N	Event	IR [a]	Adjusted HR [b] (95% CI)	*p* for Interaction
Age	30-64	Localized	74, 660	1281	3.59	1 (Ref.)	<0.0001
		Regional	22, 405	484	5.35	1.46 (1.30-1.63)	
		Distant	12, 259	302	18.29	3.20 (2.77-3.70)	
	≥65	Localized	67, 068	3484	12.48	1 (Ref.)	
		Regional	23, 251	1270	18.63	1.30 (1.21-1.39)	
		Distant	11, 857	445	42.71	1.63 (1.45-1.83)	
Sex	Male	Localized	96, 397	3540	8.21	1 (Ref.)	0.0016
		Regional	30, 606	1244	11.76	1.28 (1.10-1.38)	
		Distant	16, 241	513	28.14	1.84 (1.64-2.06)	
	Female	Localized	45, 331	1225	5.96	1 (Ref.)	
		Regional	15, 050	510	9.65	1.43 (1.28-1.59)	
		Distant	7875	234	26.88	2.47 (2.12-2.87)	
Year of diagnosis	2012-2013	Localized	34, 888	1757	7.17	1 (Ref.)	<0.0001
		Regional	12, 598	617	9.79	1.27 (1.15-1.39)	
		Distant	6263	185	22.47	1.78 (1.51-2.10)	

	2014-2015	Localized	34, 878	1388	7.37	1 (Ref.)	
		Regional	11, 576	453	9.84	1.21 (1.08-1.35)	
		Distant	6072	163	23.16	1.68 (1.41-2.00)	
	2016-2017	Localized	36, 683	1010	7.41	1 (Ref.)	
		Regional	11, 115	369	11.33	1.34 (1.18-1.51)	
		Distant	6047	187	27.89	2.10 (1.77-2.49)	
	2018-2019	Localized	35, 279	610	9.14	1 (Ref.)	
		Regional	10, 367	315	18.52	1.72 (1.50-1.98)	
		Distant	5734	212	42.76	2.79 (2.36-3.31)	
History of diabetes mellitus	No	Localized	112, 848	3512	6.80	1 (Ref.)	0.4644
		Regional	35, 949	1261	9.79	1.30 (1.22-1.40)	
		Distant	19, 066	552	25.20	2.04 (1.82-2.28)	
	Yes	Localized	28, 880	1253	10.43	1 (Ref.)	
		Regional	9707	493	16.52	1.38 (1.24-1.53)	
		Distant	5050	195	38.77	1.92 (1.63-2.26)	
History of hypertension	No	Localized	77, 758	1592	4.40	1 (Ref.)	0.0021
		Regional	25, 116	633	6.77	1.40 (1.27-1.54)	
		Distant	13, 732	313	18.99	2.38 (2.08-2.74)	
	Yes	Localized	63, 970	3173	11.57	1 (Ref.)	
		Regional	20, 540	1121	17.21	1.29 (1.2-1.39)	
		Distant	10, 384	434	41.52	1.81 (1.61-2.04)	
History of dysplipidemia	No	Localized	100, 170	3022	6.50	1 (Ref.)	0.3318
		Regional	34, 869	1196	9.59	1.31 (1.22-1.41)	
		Distant	19, 292	548	25.21	2.07 (1.85-2.31)	
	Yes	Localized	41, 558	1743	10.16	1 (Ref.)	
		Regional	10, 787	558	16.49	1.35 (1.23-1.49)	
		Distant	4824	199	38.32	1.85 (1.57-2.17)	

IR: incidence rate; HR: hazard ratio; CI: confidence interval.
a Number of cases per 1000 person-years; b Adjusted for age, sex, income, region, diabetes, hypertension, dyslipidemia, and treatment type.

Numerous cohort investigations and meta-analyses have confirmed an increased likelihood of atrial fibrillation (AF) among individuals with different malignancies [6-10]. Concerning disease stage, a meta-analysis of breast cancer patients reported no marked variation in AF occurrence between those with early and metastatic conditions [13]. Conversely, findings from a cohort study employing the SEER-Medicare database indicated that patients diagnosed at an advanced SEER stage showed a greater probability of developing AF, yielding inconsistent conclusions [14]. In the current research, the initial clinical phase of gastric cancer (GC) was significantly correlated with AF occurrence. This implies that the expanding tumor load or systemic dissemination in advanced GC may contribute to the onset of AF. Such an association supports previously proposed biological pathways, suggesting that systemic inflammation triggered by tumor-related cytokines plays a pivotal role in AF development among GC patients.

Earlier research has hypothesized that the shared predisposing factors for both GC and AF might explain the heightened AF risk in this population. Nonetheless, in this analysis, those without conventional AF risk determinants—notably younger, female, and non-hypertensive participants—exhibited a stronger connection between GC stage and AF incidence. One interpretation is that individuals already at high baseline risk (e.g., older, male, or hypertensive) may show a diminished incremental risk linked to tumor stage. With regard to sex, previous literature has reported that women with GC often present at a younger age and with more biologically aggressive tumors [15, 16]. Such sex-based biological distinctions, combined with the pro-inflammatory actions

of estrogen, might intensify systemic inflammation and thereby raise AF susceptibility among females. Regarding age, younger patients are frequently administered more aggressive therapeutic regimens involving higher-dose or multi-agent chemotherapy. While our multivariate analyses adjusted for initial therapy type, the dataset lacked details on specific drugs and dosage schedules in the CPLD. This limitation could partially explain the elevated AF incidence seen in younger individuals with advanced GC.

Treatment differences tied to cancer stage likely act as confounding influences as well. In particular, agents such as capecitabine and 5-fluorouracil, commonly used for metastatic GC, are linked to cardiovascular side effects [17]. Although coronary vasospasm and ischemia are the most documented toxicities, rhythm disorders like AF have also been observed with these medications [18-20]. To consider such cardiotoxic effects, the present study applied a multivariable Cox proportional hazards model controlling for initial treatments administered within four months (Model 5). Even after adjustment, cancer stage maintained a significant relationship with AF risk, though the magnitude was slightly lower than in Model 4, which excluded treatment adjustments. This suggests that treatment factors contributed partly, but not wholly, to the increased AF risk in advanced-stage GC.

Some previous reports have shown that AF risk peaks shortly after cancer diagnosis and becomes negligible about five years post-diagnosis [6, 7]. One meta-analysis found that cancer survivors living longer than 12 months had AF rates comparable to non-cancer populations, implying a time-dependent link [21]. Consistent with those findings, our data demonstrated a stronger stage-AF association within five years of diagnosis. This may reflect more intense inflammation and treatment-related stress early after detection. Survival and follow-up biases could also play a role: patients with severe, advanced GC (and thus higher AF risk) often have shorter survival, while early-stage survivors may have fewer medical interactions after several years. Nonetheless, even beyond five years, stage remained a significant determinant of AF, indicating that advanced GC continues to elevate AF risk irrespective of time elapsed since diagnosis.

Current guidelines advise AF screening through routine rhythm assessment for individuals aged ≥65 years [22, 23]. Common AF risk models integrate standard predictors such as age, smoking, hypertension, and coronary disease [24-26]. However, evidence remains scarce on whether these tools are suitable for cancer populations. Our findings point toward the necessity of AF monitoring in patients with advanced GC, who may face a disproportionate cardiovascular burden. Moreover, cancer stage itself could represent an independent predictor for AF, suggesting its potential inclusion in future AF risk models customized for oncology cohorts.

Despite its strengths, this study has several constraints. Data on inflammatory and metabolic biomarkers were unavailable, preventing a more granular assessment of biological mechanisms linking cancer progression to AF. Furthermore, treatment details were restricted to therapies provided during the initial four months post-diagnosis; subsequent modifications or additions could not be analyzed, leaving room for residual confounding. The ICD-O-3 code C16 used to identify GC encompasses both adenocarcinoma and gastric neuroendocrine tumors (NETs), introducing potential classification bias. Nonetheless, NETs represent only 0.3-1.8% of gastric tumors [27], making their effect minimal. Additionally, the high fatality rate in advanced GC introduces a competing risk of death, which may underestimate AF incidence. Lastly, because the study population consisted almost entirely of Korean individuals, findings may not be generalizable to other ethnic groups. Distinct epidemiological and biological profiles of GC between Asian and non-Asian populations [28-30] emphasize the need for external validation in more diverse settings.

Conclusion

Using a comprehensive nationwide database, this investigation demonstrated a clear association between GC stage and new-onset AF, with higher hazard ratios corresponding to more advanced stages. The relationship was most evident in younger, non-hypertensive, and female subgroups. These findings emphasize the clinical relevance of continuous AF surveillance in patients with advanced-stage GC, especially those lacking traditional AF risk factors.

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