Lung Cancer as a Risk Factor for Abdominal Aortic Aneurysm

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Abstract

Background

Lung cancer and abdominal aortic aneurysm (AAA) have several common risk factors. Considering that AAA is fatal, precise diagnosis and management of AAA would result in long-term survival benefit in patients with early lung cancer with good prognosis. We aimed to assess the prevalence and characteristics of AAA in patients with resectable non-small cell lung cancer (NSCLC).

Methods

Between January 2019 and November 2020, 1,019 patients with primary NSCLC treated surgically in Severance and Kangbuk Samsung Hospitals were reviewed retrospectively. We re-read abdominal-pelvic computed tomography (APCT) and positron emission tomography (PET) images and evaluated the presence of AAA. The control group comprised 2,899 cancer-free people who had a health check-up CT scan in Severance between January 2018 and December 2019. The Institutional Review Board and Ethics Committee of Severance Hospital approved this study (IRB number: 4–2021–1430).

Results

Among patients with resectable primary NSCLC patients, 39/1,019 (3.8%; odds ratio [OR], 19.19; 95% confidence interval [CI], 8.10–46.46) had AAA compared with 6/2,899 (0.2%) in the control (P < 0.001). Smokers were more likely to have AAA than non-smokers (7.0% vs 0.8%; OR, 9.57; 95% CI, 3.38–27.14; P < 0.001). In multivariable regression analysis, male sex (OR, 13.24; 95% CI, 1.50–117.48; P = 0.020), older age (OR, 1.10; 95% CI, 1.04–1.15; P < 0.001), current smoker status (OR, 4.20; 95% CI, 1.20–14.62; P = 0.024), and coronary artery obstructive disease (OR, 3.13; 95% CI, 1.48–6.62; P = 0.003) were independent risk factors for AAA development in lung cancer.

Conclusions

In our real-world study, patients with early lung cancer has a significantly higher prevalence of AAA than cancer-free controls, indicating they are a high-risk group for AAA. Therefore, we suggest patients with early NSCLC, especially smokers older than 60 years, undergo regular AAA surveillance with long term post-op follow-up for not only lung cancer but also AAA.

Background

An abdominal aortic aneurysm (AAA) is defined as dilatation of the abdominal aorta to more than 3.0 cm in diameter. Although the incidence and prevalence of AAA is low, the mortality of ruptured AAA is very high; 59–83% of patients with ruptured AAA die before they can be admitted to hospital. The mortality
rate of emergent surgery after rupture is more than 40%, and only 10–25% of them are likely to survive until discharge.[1] [2] The growth rate and risk of rupture in AAA increase proportionally to the diameter, which increases over time. Therefore, patients with AAA on initial screening are recommended to undergo regular surveillance every 6 months to 3 years, depending on the aneurysm size.[3] Because the regular surveillance and a timely intervention are important for survival in high risk patients for AAA.

Lung cancer is one of the most common cancer in worldwide and the death rate is relatively higher than other cancers. The 5-year survival rate for early stage lung cancer is 59% whereas that of advanced lung cancer is 6%.[4] The number of patients with early stage lung cancer has increased owing to early diagnosis using low-dose computed tomography (LDCT) screening.[5] Consequently, the proportion of patients with resectable lung cancer has increased, and the prognosis of lung cancer has also improved.[6]

Several risk factors of AAA, including smoking, male sex, older age, hypertension, dyslipidemia, coronary artery obstructive disease (CAOD), and chronic obstructive pulmonary disease (COPD), are also risk factors of lung cancer.[7] [8] [9]

It is unclear whether AAA surveillance is beneficial for patients with advanced lung cancer whose life expectancy is short. However, early detection and active monitoring of AAA would be beneficial for long-term survival in patients with early lung cancer because AAA rupture can be fatal, and the prognosis of early lung cancer is good.

Positron emission tomography-computed tomography (PET-CT), which includes non-contrast abdominal-pelvic computed tomography (APCT), is used for clinical staging of lung cancer. Therefore, whether AAA is present in patients with lung cancer who are candidates for curative surgery can be verified.

We aimed to examine the prevalence of AAA and its characteristics in patients with early lung cancer who were eligible for resection.

### Methods

### Patients

From January 2019 to November 2020, 1,391 patients underwent lung cancer resection at Severance Hospital and Kangbuk Samsung Hospital. Among them, 372 patients with primary non-small cell lung cancer (NSCLC) without abdominal imaging (APCT and PET-CT) or underwent palliative surgery were excluded from the analysis. A total of 1,019 patients with primary NSCLC who underwent lung cancer resection were reviewed retrospectively. Demographic and risk factor data (age, sex, smoking history, body mass index [BMI], and comorbidities such as hypertension, diabetes mellitus, dyslipidemia, COPD, CAOD, peripheral artery occlusive disease [PAOD], and chronic kidney disease [CKD]) were extracted from electronic medical records. Current smokers were defined as those who still smoked at the time of lung
cancer diagnosis, and former smokers were defined as those who stopped smoking before the diagnosis of lung cancer. Histologic types and stages of lung cancer were also analyzed.

We also obtained data from a control group consisting of people who underwent a health check-up CT scan in Severance between January 2018 and December 2019 to compare the prevalence of AAA between the lung cancer and general populations. There were a total of 2,899 participants in the cancer-free control group. Among them, we excluded those with malignancy of any type. The Institutional Review Board and Ethics Committee of Severance Hospital approved this study (IRB number: 4–2021–1430).

Measurement Of Aaa

We retrospectively re-read and measured the diameter of the abdominal aorta on all patients’ APCT and PET-CT. The maximum aneurysm diameter of the abdominal aorta derived from abdominal imaging was based on the outer wall-to-outer wall distance in the plane perpendicular to the path of the aorta. An aneurysm was defined as an aortic diameter > 3.0 cm.[10]

Statistical analysis

Continuous variables are expressed as mean ± SD and categorical variables are presented as percentage value (n/total). The comparison of the prevalence of AAA between the lung cancer and cancer-free control groups was analyzed using the Pearson χ² test. We analyzed the independent associations of multiple variables as a risk factor of AAA in patients with lung cancer using a univariable logistic regression model. Using factors that were significantly related to the development of AAA in lung cancer in the univariable logistic regression analysis, we assessed the independent risk factors of AAA in NSCLC with multivariable logistic regression analysis. Adjusted odds ratios (ORs) with associated 95% confidence intervals (CIs) were calculated. An adjusted P-value < 0.05 was considered statistically significant. All statistical analysis were performed using SPSS version 26 (SPSS, Chicago, IL, USA).

Results

Baseline characteristics

Among the 1019 patients with primary NSCLC who underwent lung resection, the mean age was 64.2 years (standard deviation 9.6 years), 56.0% (571/1,019) were male. 33.6% (342/1,019) were former smokers and 15.8% (161/1019) were current smokers. The most common cancer stage was stage I (74.2%, 756/1,019). The most common cancer type was adenocarcinoma (81.8%, 834/1,019). Table 1 gives more information about other risk factors for AAA, histologic data, and cancer stage.
## Table 1
Baseline characteristics

|                      | All patients (N = 1,019) | AAA (n = 39) | Non-AAA (n = 980) |
|----------------------|--------------------------|--------------|-------------------|
| **Age, years**       | 64.2 ± 9.6               | 70.9 ± 7.0   | 63.9 ± 9.6        |
| **Sex**              |                          |              |                   |
| Male                 | 571 (56.0)               | 38 (97.4)    | 533 (54.4)        |
| Female               | 448 (44.0)               | 1 (2.6)      | 447 (45.6)        |
| **Smoking history**  |                          |              |                   |
| Never                | 516 (50.6)               | 4 (10.3)     | 512 (52.2)        |
| Former               | 342 (33.6)               | 20 (51.3)    | 322 (32.9)        |
| Current              | 161 (15.8)               | 15 (38.5)    | 146 (14.9)        |
| **Smoking pack-years** | 19.6 ± 25.0             | 36.8 ± 20.1  | 18.3 ± 25.0       |
| **HTN**              | 479 (47.0)               | 29 (74.4)    | 450 (45.9)        |
| **DM**               | 241 (23.7)               | 12 (30.8)    | 229 (23.4)        |
| **Dyslipidemia**     | 236 (23.2)               | 14 (35.9)    | 222 (22.7)        |
| **COPD**             | 133 (13.1)               | 19 (48.7)    | 114 (11.6)        |
| **CAOD**             | 100 (9.8)                | 13 (33.3)    | 87 (8.9)          |
| **PAOD**             | 11 (1.1)                 | 1 (2.6)      | 10 (1.0)          |
| **CKD**              |                          |              |                   |
| CKD stage 1          | 364 (35.7)               | 10 (25.6)    | 354 (36.1)        |
| CKD stage 2          | 509 (50.0)               | 15 (38.5)    | 494 (50.4)        |
| CKD stage 3          | 136 (13.3)               | 12 (30.8)    | 124 (12.7)        |
| CKD stage 4          | 5 (0.5)                  | 1 (2.6)      | 4 (0.4)           |
| CKD stage 5          | 5 (0.5)                  | 1 (2.6)      | 4 (0.4)           |
| **BMI, mean ± SD**   | 24.2 ± 3.5               | 24.0 ± 2.6   | 24.2 ± 3.5        |
| Obesity (BMI ≥ 30 kg/m²) | 45 (4.4)               | 1 (2.6)      | 44 (4.5)          |
| **Cancer stage**     |                          |              |                   |
| Stage I              | 756 (74.2)               | 24 (61.5)    | 732 (74.7)        |
| Stage II             | 152 (14.9)               | 8 (20.5)     | 144 (14.7)        |
### Aaa Prevalence In Smokers (Former And Current) And Non-smokers

In total, 49.4% (503/1,019) of lung cancer patients had a positive smoking history. The mean number of smoking pack-years (PY) was 19.6 PY. A significantly higher proportion of smokers than non-smokers had AAA (7.0% [35/503] vs. 0.8% [4/516]; OR, 9.57; 95% CI, 3.38–27.14; P < 0.001; Table 2).

| AAA, abdominal aortic aneurysm; BMI, body max index; HTN, hypertension; DM, diabetes mellitus; COPD, chronic obstructive pulmonary disease; CAOD, coronary artery obstructive disease; CKD, chronic kidney disease |
|---|

Categorical variables are presented as n (%). Pearson χ² test was used to determine significant differences (P < 0.05).

Continuous variables are presented as mean ± SD. Student t-test was used to determine significant differences (P < 0.05).

#### Table 2

|   | Smoker | Non-smoker | Total |
|---|--------|------------|-------|
| AAA | 35     | 4          | 39    |
| No AAA | 468   | 512        | 980   |
| Prevalence, % (n/total) | 7.0% (35/503) | 0.8% (4/516) | 1,019 |

**OR, 9.57; 95%, CI 3.38–27.14, P < 0.001**

Values are reported as n.

Pearson χ² test was used to determine significance (P < 0.05)

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All patients

|   | All patients (N = 1,019) | AAA (n = 39) | Non-AAA (n = 980) |
|---|--------------------------|--------------|-------------------|
| Stage III | 111 (10.9) | 7 (17.9) | 104 (10.6) |
| Cancer type | | | |
| Adenocarcinoma | 834 (81.8) | 24 (61.5) | 810 (82.7) |
| Squamous carcinoma | 152 (14.9) | 13 (33.3) | 139 (14.2) |
| Other (non-small cell) | 33 (3.2) | 2 (5.1) | 31 (3.2) |
Aaa Prevalence In Lung Cancer Vs. Cancer-free Control Group

The prevalence of AAA was 3.8% (39/1,019) in the lung cancer group and 0.2% (6/2,899) in the control group. Using the Pearson $\chi^2$ test, the lung cancer group had a significantly higher prevalence of AAA than the control group (OR, 19.19; 95% CI, 8.10–46.46; P < 0.001; Table 3). In terms of AAA risk factors between the two groups, the mean age in the lung cancer group was significantly higher than that in the control group (64.2 ± 9.6 vs. 54.4 ± 10.3 years, P < 0.001; Table 3). As older age is an important risk factor for AAA development, this difference may have contributed to the higher prevalence of AAA in the lung cancer group. However, multivariable logistic regression analysis revealed that the prevalence of AAA was 10-fold higher in the lung cancer group than in the cancer-free control group, even after adjusting for age, sex, smoking history, and other AAA risk factors (Table 4), indicating that lung cancer is also an independent risk factor for AAA development.

| Table 3
Comparison of baseline characteristics and AAA prevalence between lung cancer and control groups |
|---|---|---|
| Lung cancer | Control | P value |
| Male sex | 56.0 (571/1,019) | 57.5 (1666/2,899) | 0.427 |
| Age, years | 64.2 ± 9.6 | 54.4 ± 10.3 | < 0.001 |
| Smoker | 49.4 (503/1,019) | 47.3 (1378/2,899) | 0.070 |
| Former | 33.6 (342/1,019) | 27.6 (800/2,899) |
| Current | 15.8 (161/1,019) | 19.9 (578/2,899) |
| AAA | 3.8 (39/1,019) | 0.2 (6/2,899) | < 0.001 |

Odds ratio [OR] for AAA, 19.19; 95% CI, 8.10–45.46

Categorical variables are reported as percentage (n/N).
Continuous variables (i.e., age) are reported as mean ± SD
Pearson $\chi^2$ test was used to determine significance (P < 0.05)

AAA, abdominal aortic aneurysm; OR, odds ratio; CI, confidence interval
Table 4
Univariable and multivariable logistic regression analysis of several risk factors for the presence of abdominal aortic aneurysm (AAA) in lung cancer and control groups

|                        | Univariable analysis |                          |  | Multivariable analysis |                          |  |
|------------------------|----------------------|--------------------------|  |                        |--------------------------|  |
|                        | OR                   | 95% CI for OR            | P | Adjusted               | 95% CI for adjusted      | P |
| Lung cancer            | 19.19                | 8.10                     | <0.001 | 10.78                  | 4.11                     | <0.001 |
| Sex, male              | 33.75                | 4.65                     | 0.001 | 30.75                  | 4.15                     | 0.022 |
| Age                    | 1.14                 | 1.10                     | <0.001 | 1.09                   | 1.04                     | <0.001 |
| Age ≥ 60 years         | 11.69                | 4.60                     | <0.001 |                       |                          |   |
| BMI                    | 1.02                 | 0.80                     | 0.883 |                       |                          |   |
| Smoking, PY            | 1.02                 | 1.01                     | <0.001 |                       |                          |   |
| Smoking, y/n‡          | 9.57                 | 3.38                     | <0.001 |                       |                          | <0.001 |
| Former smoker          | 9.92                 | 3.32                     | <0.001 | 6.05                   | 2.02                     | 0.001 |
| Current smoker         | 12.05                | 4.07                     | <0.001 | 15.96                  | 5.04                     | 0.022 |
| HTN                    | 7.40                 | 3.74                     | <0.001 | 2.50                   | 1.14                     | 0.096 |
| DM                     | 2.32                 | 1.19                     | 0.013 | 0.52                   | 0.24                     | 0.060 |
| Dyslipidemia           | 1.80                 | 0.98                     | 0.060 |                       |                          |   |
| CAOD                   | 12.13                | 6.21                     | <0.001 | 2.74                   | 1.21                     | 0.016 |

CI, confidence interval; OR, odds ratio; BMI, body mass index; PY, pack-years; y/n, yes/no; HTN, hypertension; DM, diabetes mellitus; CAOD, coronary artery obstructive disease

Bold P-values indicate statistical significance.

For continuous variables (age), the OR represents the change for every 1-unit change in the independent variable (years). Male sex, older age, smoking history, HTN, and CAOD were positive independent risk factors for AAA. Adjusted odds ratios are represented by the OR.

‡ Former & current smokers compared with never-smokers.

Risk factors for AAA development in patients with lung cancer
In the univariable logistic regression analysis, male sex (OR, 31.87; 95% CI, 4.36–233.03, P = 0.001), increasing age (OR, 1.10; 95% CI, 1.05–1.15, P < 0.001) and especially age > 60 years (OR, 17.33; 95% CI, 2.37–126.83), smoking history (OR, 9.57; 95% CI, 3.38–27.14, P < 0.001), smoking PY (OR, 1.02; 95% CI 1.01–1.03, P < 0.001), HTN (OR, 3.15; 95% CI 1.65–7.09, P = 0.001), COPD (OR, 7.22; 95% CI, 3.74–13.93, P < 0.001), CAOD (OR, 5.13; 95% CI, 2.55–10.35, P < 0.001), CKD stage (P = 0.004), and lung cancer pathology (squamous lung cancer, OR, 3.16; 95% CI, 1.57–6.35, P = 0.005) were independent risk factors for AAA in lung cancer (Table 4). We analyzed the independent contributing factors to higher AAA prevalence in lung cancer using multivariable logistic regression of the significant AAA risk factors identified in the univariable analysis. Because smoking history (yes/no), smoking amount (PY), and COPD have a common factor of smoking and are closely related to each other, we only used smoking history (yes/no) in the multivariable logistic regression analysis. Male sex (OR, 13.238; 95% CI, 1.492–117.482; P = 0.020), increasing age (OR, 1.10; 95% CI, 1.04–1.15; P < 0.001), smoking history (P = 0.021), and CAOD (OR, 3.13; 95% CI, 1.48–6.62; P = 0.003) were independent risk factors for AAA in lung cancer in the multivariable logistic regression model (Table 5).
Table 5
Univariable and multivariable logistic regression analysis of several risk factors for the presence of abdominal aortic aneurysm (AAA) in the lung cancer group

| Risk Factor        | Univariable analysis | Multivariable analysis |
|--------------------|----------------------|------------------------|
|                    | OR  | 95% CI for OR | P  | Adjusted OR | 95% CI for adjusted OR | P  |
| Sex, male          | 31.87 | 4.36       | 233.03 | 0.001       | 13.24 | 1.49       | 117.48 | 0.020   |
| Age†               | 1.10 | 1.05       | 1.15   | <            | 1.10 | 1.04       | 1.15   | < 0.001 |
| Age ≥ 60 yrs       | 17.33 | 2.37       | 126.83 | 0.005       |       |            |        |        |
| Obesity            | 0.56 | 0.08       | 4.17   | 0.571       |       |            |        |        |
| Smoking, PY        | 1.02 | 1.01       | 1.03   | <            | 1.10 | 1.04       | 1.15   | < 0.001 |
| Smoking, y/n‡      | 9.57 | 3.38       | 27.14  | <            |       |            |        | 0.021   |
| Former smoker      | 7.95 | 2.69       | 23.47  | <            | 1.72 | 0.52       | 5.76   |        |
| Current smoker     | 13.15 | 4.30      | 40.23  | <            | 4.20 | 1.20       | 14.62  |        |
| HTN                | 3.15 | 1.65       | 7.09   | 0.001       |       |            |        | 0.179   |
| DM                 | 1.46 | 0.73       | 2.92   | 0.289       |       |            |        |        |
| Dyslipidemia       | 1.91 | 0.98       | 3.74   | 0.058       |       |            |        |        |
| COPD               | 7.22 | 3.74       | 13.93  | <            |       |            |        | 0.001   |
| CAOD               | 5.13 | 2.55       | 10.35  | <            | 3.13 | 1.48       | 6.62   | 0.003   |
| PAOD               | 2.55 | 0.32       | 20.45  | 0.377       |       |            |        |        |
| cancer stage       |       |            |        | 0.0178      |       |            |        |        |
| CKD stage*         |       |            |        | 0.004       |       |            |        | 0.384   |
| stage2             | 1.08 | 0.48       | 2.42   | 0.862       |       |            |        |        |
| stage3             | 3.43 | 1.44       | 8.12   | 0.005       |       |            |        |        |
| stage4             | 8.85 | 0.91       | 86.49  | 0.061       |       |            |        |        |
| stage5             | 8.85 | 0.91       | 86.49  | 0.061       |       |            |        |        |
### Discussion

We observed that patients with early lung cancer had a significantly higher prevalence of AAA than that of the general population. Old age (especially > 60 years), male sex, smoking history, and CAOD were independent risk factors for AAA development in patients with lung cancer. The significant association between AAA and early lung cancer suggests a potential benefit for optimized screening for AAA in patients with lung cancer eligible for lung resection surgery.

The key pathologic characteristics of AAA include vascular inflammation, oxidative stress, destruction of the aortic extracellular matrix (ECM), and thinning of the aortic wall from loss of vascular smooth muscle cells.[11] The risk factors for AAA in lung cancer we identified would contribute in some way to the pathophysiology of AAA. Male sex is a well-known major predisposing factor (4–6 times more prevalent in men) in AAA development, consistent with our result. Previous studies found that endogenous sex hormone signalling contributed to sex differences in AAA; androgens stimulate key pathological processes in AAA, while oestrogen inhibits these processes.[12] Moreover, CAOD and AAA are closely related; the prevalence of CAOD in AAA is significantly higher than that in the general population and vice versa.[13] It is unclear whether the strong association is simply due to shared risk factors or if there are other causes beyond that.[14] Smoking is a predominant risk factor for not only lung cancer but also AAA. Smoking is known to have a positive relationship with incremental increased growth rate of AAA up to 0.4 mm per year.[15] Moreover, there is a dose-dependent relationship between smoking and AAA; it
has been shown that smoking duration and total lifetime smoking exposure both directly correlate with increased risk of AAA.[16]

In our study, AAA prevalence in smokers was 9-fold higher than that in non-smokers, which is much higher than previously reported results (2–5-fold).[17] [18] although the pattern is consistent with previous reports that smoking is an important risk factor for developing AAA.[19] [20] That might be because in our study group, patients with lung cancer were highly likely to be heavy smokers. Moreover, in our study, smoking amount showed a dose-dependent relationship with the prevalence rate of AAA in patients with lung cancer (OR, 1.02; 95% CI, 1.01–1.03; P < 0.001; Table 4), similar to previous study results.[17] Since AAA diameter increases over time, age is also a risk factor, as certain intrinsic damage to the aortic vasculature that contributes to AAA development also accumulates with advancing age in patients with lung cancer.

Although the prevalence of AAA in patients with lung cancer was significantly higher than that in our control group, it was lower than that previously reported (3.8% vs. 11.1%).[21] Even among patients with lung cancer aged > 65 years, the prevalence of AAA was 6%. This could be because AAA prevalence is lower in Asians than in Caucasians.[22] Another possible reason is that the study population of previous studies included higher proportions of patients with advanced lung cancer, squamous cell lung cancer, and small cell lung cancer, which are known to be associated with heavy smoking.[23] Furthermore, almost half of our study population were never smokers. In the univariable logistic regression model, squamous cell lung cancer was related to higher risk of AAA development than adenocarcinoma. This might be related to squamous cell lung cancer being more strongly related than adenocarcinoma to smoking.[24]

Current guidelines recommend interventional treatment (surgical or endovascular repair) only when the AAA diameter exceeds 5.5 cm. For small AAAs (3.0–5.4 cm), regular monitoring with ultrasonography or CT regularly based on its diameter is recommended.[25] Former smokers who quit smoking for > 25 years have similar relative risk of developing AAA as that of never smokers.[26] Furthermore, there is a decline in risk of AAA of approximately 30% for each decade after quitting.[17] Thus, smoking cessation is important with respect to not only lung cancer, but also AAA surveillance and reducing the growth rate of AAA.

Several studies have investigated medications for AAA aimed at reducing aortic inflammation and proteolysis and supporting vascular smooth muscle cell recovery. However, there is no strong scientific evidence that supports pharmacological treatment to reduce AAA growth in humans[27] [28]; the benefit of pharmacologic therapies, such as statins,[29] antihypertensive drugs (beta blockers and angiotensin-converting enzyme inhibitors),[30] [31] metformin,[32] and antibiotics (roxithromycin and doxycycline), [33] [34] in preventing rupture in small AAAs is controversial. However, there is some evidence that high blood pressure increases the risk of developing AAA.[35] Therefore, strict control of blood pressure in patients with lung cancer with AAA might be helpful as a preventive strategy for AAA complications.
Based on the proven cost-effective benefit of population-based AAA screening programs in high-risk groups,[2] the US Preventive Services Task Force (USPSTF) recommends screening with ultrasonography for patients at high risk of AAA (men 65 to 75 years of age with a history of smoking). A previous study found that AAA prevalence is higher in patients with lung cancer.[21] In our study, the prevalence of AAA was also significantly higher in the resectable lung cancer group, indicating that patients with lung cancer are at high risk of AAA. Unlike previous studies, we evaluated the prevalence of AAA in patients with early lung cancer, where the life expectancy is much longer, which will lead to greater benefit from timely AAA management in preventing acute emergencies and subsequent fatalities due to rupture of AAAs. In our real-world database, only 6 out of 39 patients with lung cancer with AAA were diagnosed with AAA and managed by clinicians. The majority of AAAs in patients with lung cancer (84.6%; 33/39) were ignored without risk management to prevent AAA rupture.

There are several limitations of this study. First, there was limited information on family history of vascular disease and lung cancer because our database was retrospectively analyzed. Genetic factors and family history are well-known risk factors for both lung cancer[36] and AAA.[37] Therefore, family history of lung cancer or AAA may be important risk factors for AAA in patients with lung cancer. Second, the prevalence of AAA in the cancer-free group was lower than the general prevalence of AAA in Asian populations.[22] That might be related to characteristics of our study population; the cancer-free control group were younger (mean age 54.4) than 65 years, which is the cut-off age for AAA surveillance.[38] In addition, those who voluntarily undergo regular health check-ups are likely to have a healthier lifestyle, including smoking cessation.

Conclusions

In conclusion, our finding indicates that patients with early lung cancer are a high-risk group for AAA and require AAA surveillance. Therefore, we suggest patients with early lung cancer, especially those with a smoking history and older than 60 years, are considered for regular surveillance for AAA. Furthermore, they should be educated to stop smoking and control blood pressure strictly to correct modifiable risk factors of AAA. In future studies, there is a need to evaluate the cost-effectiveness of the benefits of AAA surveillance in patients with lung cancer. In addition, further studies on medications to reduce aortic inflammation and proteolysis and enhance vascular smooth muscle cell recovery to reduce complication of AAA are required.

Abbreviations

AAA, abdominal aortic aneurysm; BMI, body max index; HTN, hypertension; DM, diabetes mellitus; COPD, chronic obstructive pulmonary disease; CAOD, coronary artery obstructive disease; PAOD, peripheral arterial occlusive disease; NSCLC, non-small cell lung cancer; SCC, squamous cell carcinoma; CKD, chronic kidney disease; CI, confidence interval; OR, odds ratio; PY, pack years; y/n, yes/no; APCT, abdominal-pelvic computed tomography; PET, positron emission tomography
Declarations

Ethics approval and consent to participate

This study was performed in accordance with the Declaration of Helsinki. This human study was approved by The Institutional Review Board and Ethics Committee of Severance Hospital – approval (IRB number: 4–2021–1430).

Consent for publication

Not applicable

Availability of data and materials

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

Competing interests

The authors declare that they have no competing interests.

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none

Authors’ contributions

Concept and design of the study by Sang Hoon Lee. Data collection and management by Hye Ran Gwon, Seong Yong Park, Jun Seong Kwon, Sang Hoon Lee. Statistical analysis and drafting of the manuscript by Hye Ran Gwon. Review and final approval of the manuscript by all the authors. All authors read and approved the final manuscript.

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