Systematic Review and Meta-Analysis

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Human immunodeficiency virus infection and mortality risk among lung cancer patients

A systematic review and meta-analysis

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Abstract

Background: Previous studies have suggested that patients with human immunodeficiency virus (HIV) infection are at higher risk of lung cancer, but the impact of HIV infection on the risk of mortality among lung cancer patients is still unclear. We conducted a systematic review and meta-analysis to clarify the association between HIV infection and mortality risk among lung cancer patients.

Methods: *PubMed* and *Embase* databases were searched to identify studies assessing the association between HIV infection and mortality risk among lung cancer patients. Only studies reporting adjusted relative risk (RR) of mortality among lung cancer patients with HIV infection were included. Meta-analysis of random-effect model was utilized to calculate the pooled RR with 95% confidence interval (CI).

Results: Twelve cohort studies were finally included. Compared with lung cancer patients without HIV infection, the pooled RR of mortality among lung cancer patients with HIV infection was 1.48 (95% CI, 1.22–1.78, $P < .001$; $I^2 = 88.6\%$). After excluding 2 studies with low quality, HIV infection was still significantly associated with an elevated risk of mortality among lung cancer patients (RR = 1.51, 95% CI, 1.25–1.82, $P < .001$; $I^2 = 89.8\%$). Sensitivity analysis showed that the statistical significance of the pooled RR was not changed by excluding any one study.

Conclusion: The outcomes from the meta-analysis provide strong evidence for the elevated risk of mortality among lung cancer patients with HIV infection, and HIV infection is an important prognostic factor in lung cancer patients.

Abbreviations: CI = confidence interval, HIV = human immunodeficiency virus, NOS = Newcastle–Ottawa Scale, NSCLC = nonsmall cell lung cancer, RR = relative risk, SCLC = small cell lung cancer.

Keywords: human immunodeficiency virus, lung cancer, meta-analysis, mortality

1. Introduction

Lung cancer is a major type of malignant disease worldwide, which is also the leading cause of cancer-related deaths in many countries.[1,2] Besides, the incidence rate of lung cancer is increasing obviously, especially in developing courtiers and areas with serious air pollutions.[2–4] Lung cancer mainly includes nonsmall cell lung cancer (NSCLC) and small cell lung cancer (SCLC), and the former one takes part of approximately 85% of all lung cancer cases.[5] At present, the pathogenesis of lung cancer is still unclear, and many factors have been found to be associated with lung cancer risk.[6–9] In the past several decades, large progresses have been achieved in both the diagnosis and the treatment lung cancer.[10,11] However, the prognosis of lung cancer cases is still not largely improved, and the risk of mortality among advanced lung cancer is relatively high.[12] Studies exploring the prognostic factors of lung cancer patients can help us appropriately predict the survival of lung cancer, which can help clinicians to screen those at high risk of premature mortality and adopt additional treatments to improve their survival time.[13]

Human immunodeficiency virus (HIV) incidence is increasing obviously in recent years, which has caused increasing harm to human health.[14–16] Some studies have found that patients with HIV infection are at higher risks of cancers than those without HIV infection.[17–21] Some studies also have suggested that patients with HIV infection are at higher risk of lung cancer, though the underlying mechanism is still poorly known.[20,22–24] Recent searches report that cancer patients with HIV infection may be associated with a poorer prognosis than those without HIV infection.[25,26] There are also several studies which are published to evaluate the correlation of HIV infection with the survival of lung cancer patients.[27–34] However, there are inconsistent findings from those published studies.[27–34] Several studies reported that HIV infection was a risk of mortality in lung cancer patients, but other studies reported that HIV infection was not associated with the prognosis of lung cancer patients. Therefore, the impact of HIV infection on the risk of mortality among lung cancer patients is still unclear. In the present study,

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we conducted a systematic review and meta-analysis to clarify the association between HIV infection and mortality risk among lung cancer patients.

2. Methods

2.1. Search strategy and study selection

PubMed and *Embase* databases were searched to identify studies assessing the association between HIV infection and mortality risk among lung cancer patients. We performed the literature search on February 20, 2017. The following search terms were used: (Lung cancer OR lung carcinoma OR NSCLC) AND (Human Immunodeficiency Virus OR Antiretroviral Therapy OR HIV OR AIDS OR HAART) AND (Survival OR prognosis OR mortality OR prognostic). No limitation was used during the literature search. The references of eligible studies were checked for additional studies which were found in the literature search. Ethics approval was waived because this study does not involve any human participants or animals.

The eligible studies in the meta-analysis were cohort studies that investigated the association between HIV infection and mortality risk among lung cancer patients. Besides, adjusted risk estimates of mortality comparing HIV(+) lung cancer patients with HIV(−) lung cancer patients, such as relative risks (RR) and hazard ratios (HR) with 95% confidence interval (CI), must be reported in the articles. Studies without data on the outcomes were excluded. Those studies without reporting adjusted risk estimates were also excluded.

2.2. Data extraction

We extracted the following information from included studies: name of the first author, year of publication, country, number of recruited participants, histological types of lung cancer, time of follow-up, variables adjusted for in the analysis, and risk estimates of mortality with 95% CIs. Data were extracted by 2 authors independently, and then were checked for accuracy by the third author.

2.3. Quality assessment

The Newcastle–Ottawa Scale (NOS) was used.[35] The NOS method used 3 domains to assess the quality of cohort studies, which included selection of lung cancer patients, the comparability between 2 groups, and the assessment of outcomes. According to the NOS method, 4 points, 2 points, and 3 points were awarded to those 3 domains, respectively. Studies with no <7 points were identified to have high quality, but those with ≤ 6 points were identified to have low quality.

2.4. Statistical methods

To decrease the impact of between-study variance on the pooled risk estimates, the random-effect meta-analysis by DerSimonian and Laird was used.[36] Between-study heterogeneity was evaluated using both Cochran Q test and I^2 statistic.[37,38] I^2 value $>50\%$ suggested high degree of heterogeneity. Subgroup analysis was conducted by histological types of lung cancer. Sensitivity analysis was conducted by omitting studies with low quality or by removing one study at a time. Publication bias was assessed using funnel plot. Egger test and Begg test were also used to further assess risk of publication bias.[39,40] Statistical analyses were conducted using Stata (Version 12.0, StataCorp, TX). A P value $<.05$ was identified as statistically significant difference.

3. Results

3.1. Characteristics of included studies

Through the literature search in *PubMed* and *Embase* databases, we found a total of 923 individual records, but only 22 studies were considered possibly eligible and were evaluated by reading the full articles.[7,23,27–34,41–52] Ten studies were excluded because they did not assess the impact of HIV infection on the survival of lung cancer patients or they did not report data on the estimates of mortality risk associated with HIV infection.[7,23,45–52] At last, 12 studies were included into the meta-analysis.

Table 1 presented a brief description of these 12 studies (Table 1). All studies were from North-America, and only 1 study was from Europe (Table 1). All studies reported adjusted risk estimate, and the factors used in the adjustment analyses were various among those 12 studies (Table 1). The number of recruited participants was also various among those 12 studies, ranging from 29 to 328,924 (Table 1). In addition, the number of recruited lung cancer participants with HIV infection ranged from 13 to 1058 (Table 1). The time of follow-up among those 12 studies ranged from 1 to 5 years. Among those 12 studies, only 3 studies assessed the prognostic role of HIV infection among NSCLC patients, and the other 9 studies did not report outcomes stratified by histological types of lung cancer (Table 1). The quality of included studies was assessed by NOS method, and 10 studies had scored ≥ 7 points and thus had high quality. Only studies scored 6 or fewer points had low quality (Table 1).

3.2. Meta-analysis

Heterogeneity among those 12 studies was high ($I^2 = 88.6\%$, $P < .001$). In the meta-analysis of total included studies, compared with lung cancer patients without HIV infection, the pooled RR of mortality among lung cancer patients with HIV infection was 1.48 (95% CI, 1.22–1.78, $P < .001$; Fig. 1). In the subgroup analysis by histological types, there was no significant heterogeneity among those 3 studies involving NSCLC patients ($I^2 = 39.0\%$, $P = .194$), and HIV infection was significantly associated with elevated risk of mortality among NSCLC patients (RR = 1.69, 95% CI, 1.48–1.94, $P < .001$; Fig. 2).

In the sensitivity analysis, after excluding 2 studies with low quality, HIV infection was still significantly associated with an elevated risk of mortality among lung cancer patients (RR = 1.51, 95% CI, 1.25–1.82, $P < .001$; $I^2 = 89.8\%$; Fig. 3). Bedsides, when we carried out sensitivity analyses by removing one study at a time, the statistical significance of the pooled RR was not changed by excluding any one study (Fig. 4).

3.3. Risk of publication bias

Funnel plot did not suggest risk of publication bias (Fig. 5). In addition, both Egger test and Begg test further validated the finding above, and the P values were .63 and .42, respectively.

4. Discussion

The increased risk of cancers in HIV patients has been well defined.[53–55] Previous studies have provided strong epidemiological evidence for HIV infection as risk factors of several types

Table 1

Studies on the impact of HIV infection on mortality risk among lung cancer patients.

Study	Design	Country	Participants	Follow-up	Adjusted factors	Quality
Marcus (2015)[27]	Cohort	USA	80 HIV positive lung cancer patients and 507 HIV negative lung cancer patients	5 y	Age, ethnicity, sex, TNM stage, cancer treatment type, and smoking.	High
Hessol (2015)[41]	Cohort	USA	56 lung cancer patients	Unclear	Cohort source, geographic location, calendar period, black race, ever-injected drugs, and histologic type.	Low
Coghill (2015)[28]	Cohort	USA	1,058 HIV+ lung cancer patients and 327,866 HIV− lung cancer patients	>5 y	Age at cancer diagnosis, sex, year of cancer diagnosis, ethnicity, and cancer stage at diagnosis.	High
Maso (2014)[42]	Cohort	Italy	46 HIV+ lung cancer patients and 276 HIV− lung cancer patients	5 y	Matching variables and age at diagnosis.	High
Sigel (2013)[30]	Cohort	USA	267 HIV-infected patients and 1428 similar controls with no evidence of HIV diagnosed with NSCLC	5 y	Year of cancer diagnosis, age, sex, ethnicity, marital status, median income in zip code area of residence, reason for medicare entitlement, comorbidity score, cancer stage, histologic subtype, use of stage-appropriate treatment, nursing home residence, and use of home medical services.	High
Lee (2013)[43]	Cohort	USA	174 HIV-infected patients and 3480 non-HIV NSCLC patients	5 y	Number of comorbidities, residence in a large metropolitan area, and marital status.	High
Suneja (2013)[29]	Cohort	USA	337 HIV-infected patients and 156,593 non-HIV NSCLC patients	>5 y	Year of diagnosis, age at diagnosis, sex, race/ethnicity, cancer stage, and histologic subtype, and cancer stage.	High
Hooker (2012)[44]	Cohort	USA	22 HIV-infected lung cancer patients to 2430 lung cancer patients with HIV-unspecified status	5 y	Age at diagnosis, sex, race, stage, NSCLC histologic subtype, date of surgery, and surgical procedure.	High
Rengan R 2012[31]	Cohort	USA	322 NSCLC patients with HIV group from 71,976 patients with NSCLC	>5 y	Year of cancer diagnosis, age, sex, ethnicity, comorbidity, cancer stage, tumor histologic subtype, and so on.	High
Shiels (2010)[32]	Cohort	USA	13 HIV-infected lung cancer patients and 16 HIV-uninfected lung cancer patients	>1 y	Age at diagnosis, gender, distant cancer stage, average packs smoked per day, current injection drug use at diagnosis, and insurance status at diagnosis.	Low
Brock (2006)[33]	Cohort	USA	92 HIV-infected lung cancer patients and 4973 HIV-indeterminate lung cancer patients	>1 y	Race, stage, sex, age, histologic findings, and smoking.	High
Tammemagi (2003)[34]	Cohort	USA	1155 lung cancer patients	819 d	Race, stage, sex, age, histologic findings, and smoking.	High

HIV = human immunodeficiency virus, NSCLC = nonsmall cell lung cancer.

of cancers including lung cancer.[55,56] Besides the impact of HIV infection on the prognosis of cancer patients has gained increasing concerns in recent years. There are also a number of epidemiological studies published to assess the impact of HIV infection on the survival of lung cancer patients, but no consistent findings are available. Therefore, there is a need to perform a systematic review and meta-analysis to those published studies to clarify the association between HIV infection and mortality risk among lung cancer patients. The meta-analysis will help to adequately assess the prognostic role of HIV infection in lung cancer patients.

To our knowledge, the present study is the first meta-analysis of studies on the association between HIV infection and mortality risk among lung cancer patients. A total of 12 studies were included and the pooled outcomes confirmed an important prognostic role of HIV infection among lung cancer patients (Fig. 1). Moreover, the consistent outcomes from the subgroup analyses and sensitivity analyses added the epidemiological evidence for the adverse influence of HIV infection in lung cancer patients. Therefore, this study provides strong evidence for the elevated risk of mortality among lung cancer patients with HIV infection, and HIV infection is an important prognostic factor of lung cancer patients.

The biological mechanisms underlying the association between HIV infection and increased risk of mortality among lung cancer patients are unclear at present. HIV infection is associated with poor immune regulatory function, which may make the immune system in lung cancer patients lose the ability to control the proliferation or metastasis of tumor cells.[57,58] It has also been suggested that the respiratory system is particularly susceptible to the damages caused by HIV-related immunosuppression.[59] Some infectious and noninfectious pulmonary diseases are also associated with lung cancer patients, and since HIV-related immunosuppression can result in infectious and noninfectious pulmonary diseases, and HIV infection can thus promote the progression of lung cancer. However, the molecular mechanisms explaining the increased risk of mortality among lung cancer patients with HIV infection are still poorly defined, and more future studies are recommended to provide new insights into it.

There were several strengths in the study. First, the total number of included participants was large and could lead to a

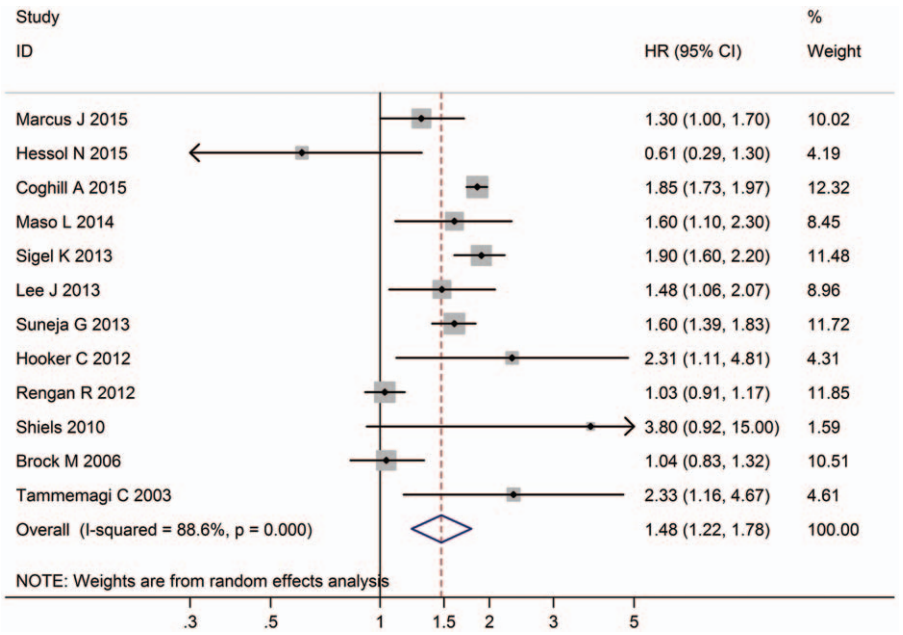

Figure 1. Meta-analysis of total 12 studies suggested an elevated risk of mortality among lung cancer patients with HIV infection.

correct estimate of the role of HIV infection in lung cancer patients. Second, the consistent outcomes in the sensitivity analyses proved the credibility of the pooled outcomes. Finally, all included studies provided adjusted RRs which had controlled the impact of other confounding factors on the association between HIV infection and prognosis of lung cancer patients. The pooled outcomes based on adjusted risk estimates showed that HIV infection was an independent risk factor of mortality in lung cancer patients.

The present meta-analysis also had several limitations. First, the test for heterogeneity among those included studies was significant ($I^2 = 88.6\%$, $P < .001$). The obvious heterogeneity may be caused by the variance in study design, characteristics of lung cancer patients, the different treatment methods of HIV

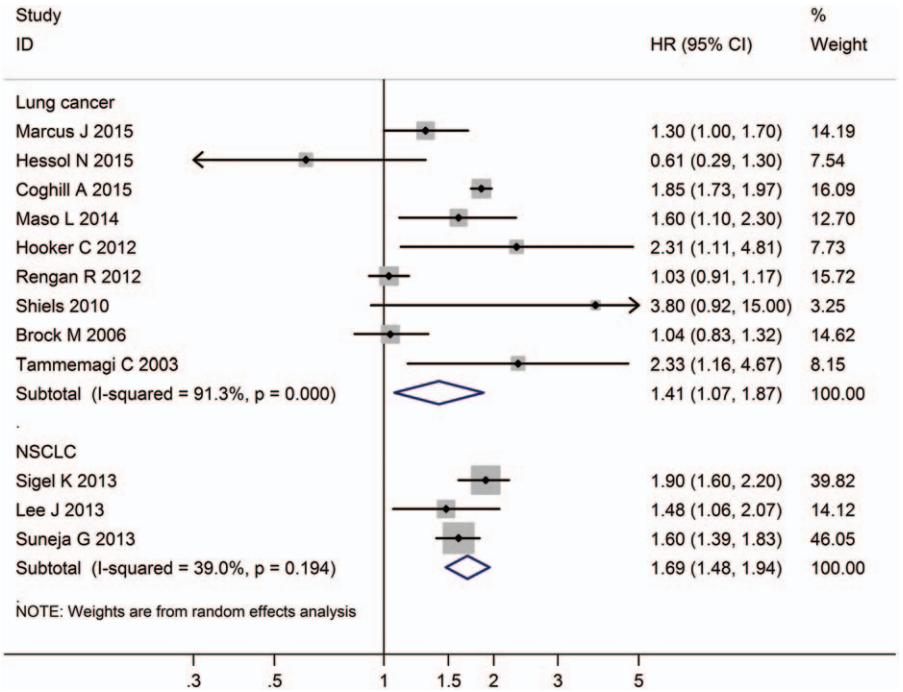

Figure 2. Forest plot in the subgroup analysis stratified by histological type of lung cancer.

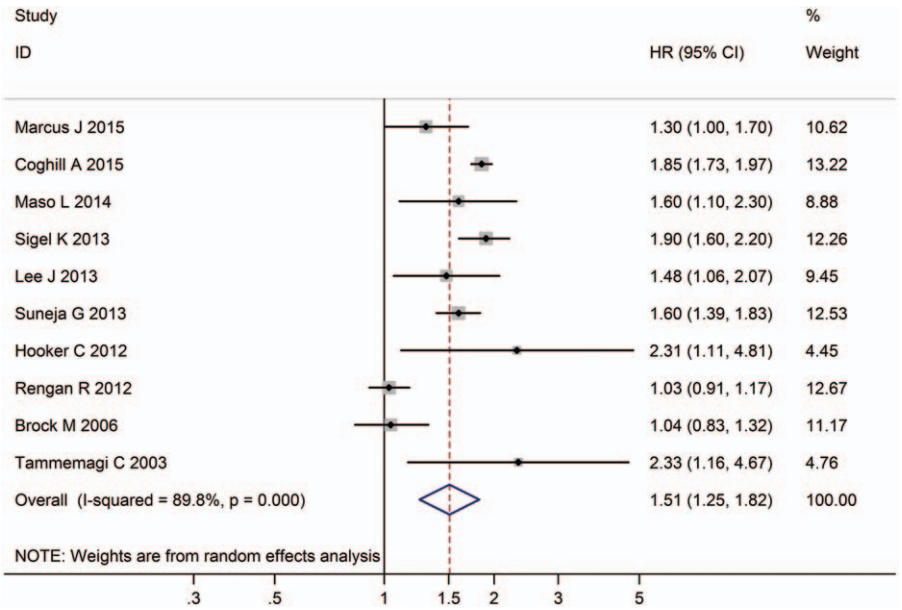

Figure 3. Forest plot in the meta-analysis of 10 studies with high quality.

infection, or the different treatment methods of lung cancer. Future studies are needed to explore whether those factors are the sources of heterogeneity, and to assess whether obvious differences exist in the association between HIV infection and the survival of lung cancer patients with different stages or different treatments. Second, only 3 studies reported outcomes of interest in NSCLC patients, and no study assessed the prognostic role of HIV infection on SCLC patients. Therefore, more studies are needed to explore the prognostic role of HIV infection in SCLC patients. Finally, some of those studies were retrospective cohort studies, and were likely to be affected by some biases, such as selection bias. More prospective cohort studies are needed to provide a more appropriate evaluation of the role of HIV in lung cancer patients.

In conclusion, this study provides strong evidence for the elevated risk of mortality among lung cancer patients with HIV infection. Future studies are recommended to assess the prognostic role of HIV infection in SCLC patients or lung cancer

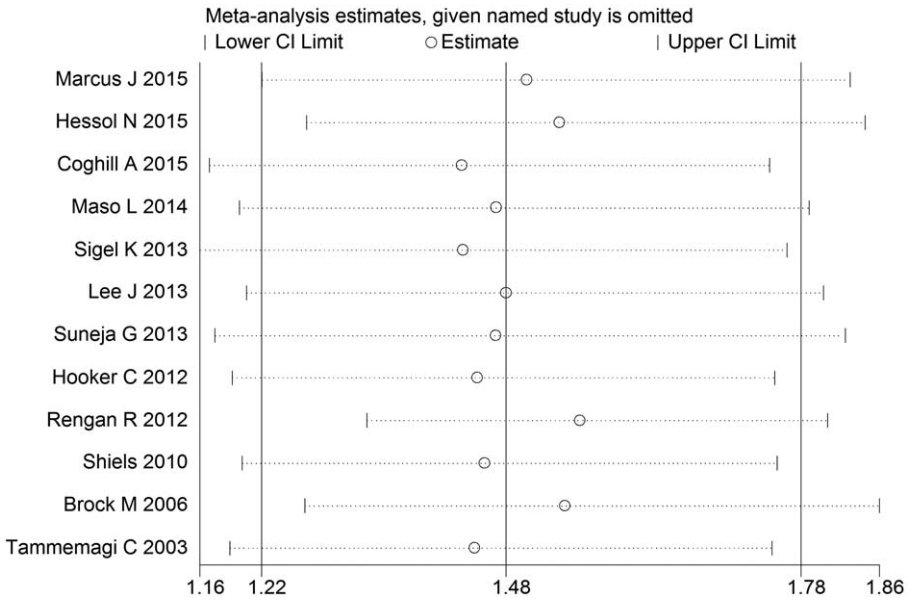

Figure 4. Forest plot showed the outcomes of sensitivity analyses by removing one study at a time.

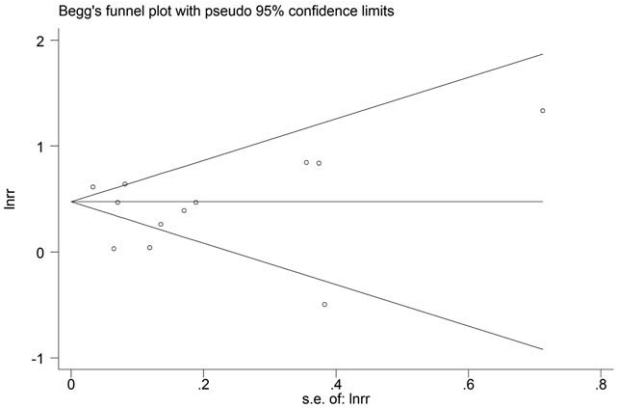

Figure 5. Funnel plot in the meta-analysis of total 12 included studies.

patients from Asian or African populations. Besides, more researches are also needed to clarify the potential underlying mechanisms.

Author contributions

Conceptualization: X.-D. Shen, Y.-H. Wang.
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Formal analysis: X.-D. Shen. Y.-H. Wang.
Writing – original draft: X.-D. Shen, Y.-H. Wang.
Writing – review & editing: X.-D. Shen.

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