Observational Study

Medicine®

OPEN

Pneumoconiosis increases the risk of congestive heart failure

A nationwide population-based cohort study

Chia-Ming Yen (MD)[a], Cheng-Li Lin (MSc)[b,c], Ming-Chia Lin (PhD)[d], Huei-Yong Chen (MD)[d], Nan-Han Lu (MD, PhD)[e], Chia-Hung Kao (MD)[f,g,*]

Abstract

The purpose of the study was to determine the relationship between pneumoconiosis and congestive heart failure (CHF).

We collected data from the National Health Insurance Research Database in Taiwan. The study sample comprised 8923 patients with pneumoconiosis and 35,692 nonpneumoconiosis controls enrolled from 2000 to 2011. Patients were followed up until the end of 2011 to evaluate the incidence of CHF. The risk of CHF was analyzed using Cox proportional hazard regression models, and the analysis accounted for factors such as sex, age, comorbidities, and air pollutants ($\mu g/m^3$).

The overall incidence of CHF was higher in the pneumoconiosis cohort (15.7 per 1000 person-y) than in the nonpneumoconiosis cohort (11.2 per 1000 person-y), with a crude hazard ratio (HR) of 1.40 ($P < 0.001$). The HR for CHF was 1.38-fold greater in the pneumoconiosis cohort than in the nonpneumoconiosis cohort ($P < 0.001$) after the model was adjusted for age, sex, various comorbidities, and air pollutants ($\mu g/m^3$). The relative risk for CHF in the sex-specific pneumoconiosis cohort compared with the nonpneumoconiosis cohort was significant for men (adjusted HR = 1.40, 95% confidence interval = 1.21–1.62, $P < 0.001$). The incidence density rates of CHF increased with age; pneumoconiosis patients had a higher relative risk of CHF for all age group.

Patients with pneumoconiosis were at higher risk for developing CHF than patients in the nonpneumoconiosis cohort, particularly in cases with coexisting coronary artery disease, hypertension, and chronic obstructive pulmonary disease.

Abbreviations: CHF = congestive heart failure, CI = confidence interval, HR = hazard ratio, ICD-9-CM = International Classification of Diseases, Ninth Revision, Clinical Modification, LHID = Longitudinal Health Insurance Database, NHI = National Health Insurance, NHIRD = National Health Insurance Research Database, PM = particular matter.

Keywords: air pollutants, congestive heart failure, National Health Insurance, nationwide population-based cohort study, pneumoconiosis

1. Introduction

Pneumoconiosis, a progressive and irreversible lung disease, is a systemic occupational disease caused by long-term dust inhalation.[1,2] Patients with pneumoconiosis not only lose their livelihood, but also have a shorter life expectancy.[3] Although environmental factors are prominent in the pathogenesis of pneumoconiosis, a genetic component of susceptibility has also been established.[4] Pulmonary disorders caused by exposure to occupational hazards and silica are typical forms of pneumoconiosis.[5] Most genetic association studies have focused on interleukin 1 and tumor necrosis factor gene families;

Editor: Feola Mauro.

M-CL and C-MY equally contributed to this work.

Authorship: Conception/design—C-MY, M-CL, and C-HK; provision of study materials—C-LL and C-HK; collection and/or assembly of data—C-MY, M-CL, and C-HK; data analysis and interpretation—C-MY, C-LL, M-CL, and C-HK; manuscript writing—all authors; final approval of manuscript—all authors.

Funding: This study is supported in part by Taiwan Ministry of Health and Welfare Clinical Trial and Research Center of Excellence (MOHW105-TDU-B-212-133019), China Medical University Hospital, Academia Sinica Taiwan Biobank Stroke Biosignature Project (BM10501010037), NRPB Stroke Clinical Trial Consortium (MOST 104-2325-B-039-005), Tseng-Lien Lin Foundation, Taichung, Taiwan, Taiwan Brain Disease Foundation, Taipei, Taiwan, and Katsuzo and Kiyo Aoshima Memorial Funds, Japan; and CMU under the Aim for Top University Plan of the Ministry of Education, Taiwan. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. No additional external funding received for this study.

The authors have no conflicts of interest to disclose.

[a] Department of Anesthesiology, Buddhist Tzu Chi General Hospital, Taichung Branch, [b] Management Office for Health Data, China Medical University Hospital, [c] College of Medicine, China Medical University, Taichung, [d] Department of Nuclear Medicine, E-DA Hospital, I-Shou University, Kaohsiung, [e] Department of Radiology, E-DA Hospital, I-Shou University, Kaohsiung, [f] Department of Nuclear Medicine PET Center, China Medical University Hospital, [g] Graduate Institute of Clinical Medical Science and School of Medicine, College of Medicine, China Medical University, Taichung, Taiwan.

[*] Correspondence: Chia-Hung Kao, Graduate Institute of Clinical Medical Science and School of Medicine, College of Medicine, China Medical University, No. 2, Yuh-Der Road, Taichung 404, Taiwan (e-mail: d10040@mail.cmuh.org.tw).

Medicine (2016) 95:25(e3972)

Received: 27 August 2015 / Received in final form: 17 May 2016 / Accepted: 26 May 2016

http://dx.doi.org/10.1097/MD.0000000000003972

however, chemokines, human leukocyte antigens, and antioxidant gene variations have also been examined.[4] Cytokines influence various biological events such as inflammation, metabolism, cell growth and proliferation, morphogenesis, fibrosis, and homeostasis.[6]

Heart failure is a clinical syndrome in which an abnormality in cardiac structure or function renders the heart unable to eject or fill with blood at a rate commensurate with the requirements of the metabolizing tissues.[7] Despite numerous advances in the field of medicine, management of congestive heart failure (CHF) has been difficult in clinical practice, and mortality rates remain high.[8] CHF can have a genetic origin or be caused by various pathological processes such as hypertension, myocardial infarction, cardiac hypertrophy, myocardial infection, ischemic heart disease, valvular defects, and alcohol misuse.[9]

Previous studies have indicated an association between air pollution and hospital admission for CHF.[10] Previous studies have provided evidence of an association between air pollution and hospital admission for CHF.[10] In Taiwan, the study provides evidence that higher levels of particular matter (PM) 2.5 increase the risk of hospital admissions for CHF.[34] To know the relationship between pneumoconiosis and CHF, we analyzed data from the National Health Insurance Research Database (NHIRD) in Taiwan.

2. Methods

2.1. Data source

The NHIRD is an administrative database containing claims records from the Taiwan National Health Insurance (NHI) program, which was implemented by the government in March 1995. The NHI program provides a comprehensive, unified, and universal health insurance program for all residents of Taiwan. The NHI program covers >99% of the 23.74 million people living in Taiwan.[11] The NHIRD was compiled and is maintained by the Department of Health and the National Health Research Institutes (NHRI) of Taiwan. The NHRI encrypts patient information to maintain privacy, and provides researchers with identification numbers for linking claims data, including patient sex, date of birth, medical services received, and prescriptions. Under the NHI program, enrollees with any catastrophic illness in 1 of 30 categories specified by the Bureau of NHI can apply for a catastrophic illness certificate, the records of which are stored in the Registry of Catastrophic Illnesses Patient Database (RCIPD). Approved applicants are exempted from copayments for medical care related to their catastrophic illness. The issuance of certificates is validated through the careful review of medical records, laboratory studies, and imaging studies by at least 2 specialists. In this study, we used the Longitudinal Health Insurance Database 2000 (LHID2000), which is a data set derived from the NHIRD containing 1 million randomly sampled beneficiaries enrolled in the NHI program in 2000, and collected all records for this population from 1996 to 2011. The NHRI states that no statistical differences in age, sex, or healthcare costs exist between the LHID2000 and all enrollees. Outpatient and inpatient disease diagnoses are coded according to the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM). This study was approved to fulfill the condition for exemption by the Institutional Review Board (IRB) of China Medical University (CMUH-104-REC2-115). The IRB also specifically waived the consent requirement.

2.2. Sampled participants

This retrospective cohort study applied data from the RCIPD and LHID2000 from 2000 to 2011. Patients with newly diagnosed pneumoconiosis (ICD-9-CM 500, 501, 502, 503, 505) between 2000 and 2011 in the RCIPD were assigned to the pneumoconiosis cohort. The date of application for a catastrophic illness certificate in approved pneumoconiosis cases was considered the index date.

Patients were excluded if they had a history of CHF (ICD-9-CM code 428) before the index date, or if their age or sex information was incomplete. Patients with no history of pneumoconiosis were randomly selected from the LHID2000. For each pneumoconiosis case, patients were 1:4 frequency-matched according to sex, age (per 5 year), and index year. The same exclusion criteria were applied to a nonpneumoconiosis cohort. We used the method of 1:4 matching to increase the statistical power and control the potential confounding (including age and sex). Finally, 8923 patients with pneumoconiosis and 35,692 controls without pneumoconiosis were included in this study.

2.3. Outcome measurement, comorbidities, and pollutant-exposure values

The main outcome was the development of CHF during the follow-up period. The person-years of the follow-up period were calculated from the index date until CHF diagnosis or censoring because of death, loss to follow-up, withdrawal from the insurance program, or December 31, 2011. Baseline comorbidities considered in this study were diabetes (ICD-9-CM code 250), hypertension (ICD-9-CM codes 401–405), hyperlipidemia (ICD-9-CM code 272), coronary artery disease (CAD) (ICD-9-CM codes 410–414), stroke (ICD-9-CM codes 430–438), and chronic obstructive pulmonary disease (COPD) (ICD-9-CM codes 491, 492, and 496). We used the data from Taiwan Environmental Protection Administration in which Taiwan's air quality data are maintained. Yearly average concentrations of air pollutants (daily PM2.5 and PM10) were calculated from January 1, 2000 to December 31, 2011.

2.4. Statistical analysis

We used the χ^2 test to determine the differences in categorical demographic variables and comorbidities between the pneumoconiosis and nonpneumoconiosis cohorts, and used the Student t test to examine the differences in mean age, PM2.5 yearly average, and PM10 yearly average between both cohorts. We assessed the cumulative incidence of CHF in both cohorts by using the Kaplan-Meier method, and estimated the differences between the 2 cohorts by using the log-rank test. The overall as well as sex-, age-, comorbidity-, and follow-up time-specific incidence density rates (per 1000 person-y) were calculated for each group. Univariate and multivariate Cox proportional hazard regression analyses were performed to estimate the hazard ratios (HRs) and 95% confidence intervals (CIs) for CHF in the sex-specific pneumoconiosis cohort compared with the nonpneumoconiosis cohort. The multivariate model was simultaneously adjusted for sex, age, comorbidities (diabetes, hypertension, hyperlipidemia, CAD, stroke, and COPD), PM2.5 yearly average, and PM10 yearly average. We used SAS Version 9.2 for Windows (SAS Institute Inc, Cary, NC) for all data analyses, and Kaplan-Meier curves were plotted using R Version 2.14.1 (R Development Core Team, Vienna, Austria). Results were considered statistically significant at $P < 0.05$.

3. Results

Table 1 shows the demographic data and comorbidities of patients in the pneumoconiosis and nonpneumoconiosis cohorts. The pneumoconiosis cohort and nonpneumoconiosis cohort exhibited no significant difference in age or sex distributions. Among the pneumoconiosis patients, 82.2% were >60 years and 86.4% were men. The mean age of the pneumoconiosis cohort and nonpneumoconiosis cohort was 66.5 (±7.45) and 66.1 (±8.02) years, respectively. The PM2.5 yearly average of 35.4 $\mu g/m^3$ for the pneumoconiosis cohort was >28.4 $\mu g/m^3$ for the nonpneumoconiosis cohort, respectively. The PM10 yearly average of 60.0 $\mu g/m^3$ for the pneumoconiosis cohort was >50.0 $\mu g/m^3$ for the nonpneumoconiosis cohort, respectively. Nonpneumoconiosis patients exhibited a higher prevalence of comorbidities (diabetes, hypertension, and hyperlipidemia, but not COPD) than the pneumoconiosis cohort ($P < .001$). After 12 years of follow-up, the cumulative incidence of CHF in the pneumoconiosis cohort was approximately 4.5% higher than that in the nonpneumoconiosis cohort ($P < .001$; Fig. 1). During 71,677 and 310,980 person-years of follow-up, the overall incidence density of CHF was significantly higher in the pneumoconiosis cohort than in the nonpneumoconiosis cohort (15.7 vs 11.2 per 1000 person-y), with a crude HR of 1.40 (95% CI = 1.31–1.50) (Table 2).

After adjustment for age, sex, comorbidities (diabetes, hypertension, hyperlipidemia, CAD, stroke, and COPD), PM2.5 yearly average, and PM10 yearly average, the pneumoconiosis patients had a 1.38-fold greater risk of CHF (95% CI = 1.21–1.58) than the nonpneumoconiosis patients. The sex-specific incidence rate of CHF was slightly higher for the women in both cohorts. The relative risk of CHF in the sex-specific pneumoconiosis cohort compared with the nonpneumoconiosis cohort was significant for men (adjusted HR = 1.40, 95% CI = 1.21–1.62). When stratified by age, the incidence density rates of CHF increased with age in both cohorts, and pneumoconiosis patients had a higher relative risk of CHF for all age group than patients without pneumoconiosis. We analyzed the association between pneumoconiosis and the risk of CHF by stratifying the comorbidities, and found a similar increased level of risk for CHF in patients with no comorbidity (adjusted HR = 1.38, 95% CI = 1.03–1.86) and those with comorbidities (adjusted HR = 1.22, 95% CI = 1.06–1.40). Table 3 shows the results of univariate and multivariate Cox proportional hazard models for the association between pneumoconiosis and CHF. The risk of developing CHF increased 1.05-fold (95% CI = 1.04–1.06) with age (per year), and the risk was higher for patients with hypertension (adjusted HR = 1.65, 95% CI = 1.47–1.86), CAD (adjusted HR = 1.68, 95% CI = 1.47–1.86), COPD (adjusted HR = 1.17, 95% CI = 1.02–1.33), and increasing 1.00-fold (95% CI = 1.00–1.01) with PM2.5 yearly average ($\mu g/m^3$). Figure 2 depicts the joint effect of pneumoconiosis and hypertension or CAD or COPD on CHF risk. A higher risk of CHF was observed in patients with pneumoconiosis and hypertension (adjusted HR = 2.78, 95% CI = 2.34–3.31), pneumoconiosis and CAD (adjusted HR = 3.00, 95% CI = 2.43–3.70), or pneumoconiosis and COPD (adjusted HR = 1.66, 95% CI = 1.42–1.94) than patients without pneumoconiosis, hypertension, CAD, or COPD.

Table 1

Characteristics of patients with pneumoconiosis and patients without pneumoconiosis.

	Pneumoconiosis				
	Yes (N = 8923)		No (N = 35,692)		
	n	%	n	%	*P*
Age, y					0.99
≤59	1587	(17.8)	6372	(17.9)	
60–69	4632	(51.9)	18,504	(51.8)	
≥70	2704	(30.3)	10,816	(30.3)	
Mean (SD)*	66.5	(7.45)	66.1	(8.02)	<0.001
Sex					0.99
Female	1218	(13.7)	4872	(13.7)	
Male	7705	(86.4)	30,820	(86.4)	
Air pollutants, $\mu g/m^3$					
PM2.5 yearly average (SD)*	35.4	(8.47)	28.4	(3.57)	<0.001
PM10 yearly average (SD)*	60.0	(13.3)	50.0	(5.41)	<0.001
Comorbidity					
Diabetes	582	(6.52)	3136	(8.79)	<0.001
Hypertension	3775	(42.3)	16,039	(44.9)	<0.001
Hyperlipidemia	1405	(15.8)	6564	(18.4)	<0.001
Coronary artery disease	1843	(20.7)	7253	(20.3)	0.48
Stroke	429	(4.81)	1899	(5.32)	0.05
COPD	4679	(52.4)	4168	(11.7)	<0.001

COPD = chronic obstructive pulmonary disease, SD = standard deviation.
χ^2 test;
* *t* test.

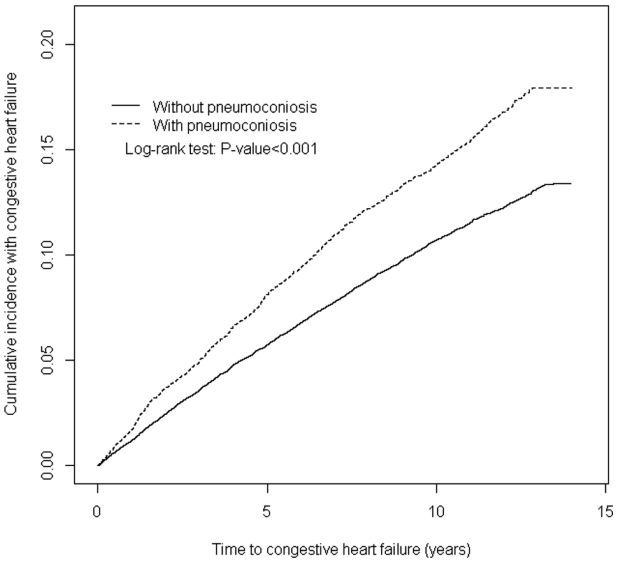

Figure 1. Cumulative incidence of congestive heart failure compared between with and without pneumoconiosis.

4. Discussion

Since 1954, pneumoconiosis was the oldest and most well-known occupational lung disease in Taiwan.[23] Even coal-mining decreased gradually in the end of the 19th century; coal-worker pneumoconiosis (CWP) remained the most prevalent type of pneumoconiosis. There are still many patients, but few studies have assessed the development and progression in this particular population.[24]

In Taiwan, the relationship between pneumoconiosis and CHF remains unclear. We hypothesized that pneumoconiosis increases the risk of CHF. In this study, the data were collected from the RCIPD and LHID2000, data sets derived from the NHIRD. To test this hypothesis, we examined 8923 pneumoconiosis cases, comparing them with 35,692 nonpneumoconiosis cases. We also included medical records in the analysis to ascertain the baseline

Table 2

Incidence and HR of congestive heart failure between patients with pneumoconiosis and without pneumoconiosis.

	Pneumoconiosis							
	Yes			No				
Outcome	Event	PY	Rate*	Event	PY	Rate*	Crude HR (95% CI)†	Adjusted HR (95% CI)‡
All	1125	71,677	15.7	3469	31,0980	11.2	1.40 (1.31, 1.50)#	1.38 (1.21, 1.58)#
Sex								
Female	187	10,924	17.1	540	44,387	12.2	1.40 (1.19, 1.66)#	1.26 (0.88, 1.80)
Male	938	60,753	15.4	2929	266,593	11.0	1.40 (1.30, 1.51)#	1.40 (1.21, 1.62)#
Age, y								
≤59	139	14,391	9.66	296	63,368	4.67	2.06 (1.69, 2.52)#	1.70 (1.09, 2.64)‖
60–69	576	40,217	14.3	1700	171,652	9.90	1.44 (1.31, 1.59)#	1.31 (1.09, 1.59)¶
≥70	410	17,069	24.0	1473	14,391	19.4	1.23 (1.10, 1.37)#	1.30 (1.04, 1.62)‖
Comorbidity§								
No	171	19,671	8.69	919	144,633	6.35	1.37 (1.16, 1.61)#	1.38 (1.03, 1.86)‖
Yes	954	52,006	18.3	2550	166,346	15.3	1.19 (1.11, 1.28)#	1.22 (1.06, 1.40)¶

CI = confidence interval, COPD = chronic obstructive pulmonary disease, PM = particular matter, PY = person-years.
* Rate, incidence rate per 1000 person-years.
† Crude HR, relative hazard ratio.
‡ Adjusted HR, hazard ratio adjusted for age, sex, and comorbidities of diabetes, hypertension, hyperlipidemia, coronary artery disease, stroke, COPD, and PM2.5 yearly average and PM10 yearly average.
§ Comorbidity: patients with any one of the comorbidities diabetes, hypertension, hyperlipidemia, coronary artery disease, stroke, and COPD were classified as the comorbidity group.
‖ $P < 0.05$.
¶ $P < 0.01$.
# $P < 0.001$.

comorbidities, namely diabetes, hypertension, hyperlipidemia, CAD, stroke, and COPD.

Differences in categorical demographic variables and comorbidities between the 2 cohorts were compared using the χ^2 test, differences in mean age between the 2 cohorts were examined using the Student *t* test, and the cumulative incidence of CHF in the 2 cohorts was assessed using the Kaplan-Meier method, the differences between which were estimated using the long-rank test.

Pneumoconiosis was found in 7.5% of the workers. The highest prevalence was found among furnace workers (15.9%) and molding workers (8.4%). The current study found that pneumoconiosis may be due to airborne concentration and high proportions of free silica and long length of exposure.[25]

The demographic data revealed that most pneumoconiosis patients were >60 years (82.2%) and were men (86.4%). Factors that increased the risk for pneumoconiosis included the concentration of respirable coal dust, coal dust size and composition, age, work environment, and work practices.[1] From other studies, we found that the incidence of pneumoconiosis increases with the length of exposure.[12]

Over the past 30 years, the prevalence and incidence of heart failure have increased markedly with age even approximately 5-fold from the age of 40 to 70 years over this period.[31] Although there have been limited data regarding the epidemiology of heart failure in Taiwan, the increase prevalence of heart failure may parallel with that seen in many western countries.[32] In Taiwan 2011, the prevalence of heart failure was also increased with age (≤59 years: 2.41%, 60–69 years: 17.31%, ≥70 years: 58.41%).

Table 3

Cox model with HRs and 95% CIs of congestive heart failure associated with pneumoconiosis and covariates.

	Crude*		Adjusted†	
Variable	HR	(95% CI)	HR	(95% CI)
Age, y	1.06	(1.06, 1.07)‖	1.05	(1.04, 1.06)‖
Sex (male vs female)	1.11	(1.03, 1.21)§	0.89	(0.77, 1.03)
Baseline comorbidities (yes vs no)				
Pneumoconiosis	1.40	(1.31, 1.50)‖	1.38	(1.21, 1.58)‖
Diabetes	1.50	(1.36, 1.65)‖	1.06	(0.89, 1.27)
Hypertension	2.14	(2.02, 2.28)‖	1.65	(1.47, 1.86)‖
Hyperlipidemia	1.40	(1.31, 1.50)‖	1.11	(0.98, 1.26)
Coronary artery disease	2.50	(2.35, 2.65)‖	1.68	(1.50, 1.89)‖
Stroke	1.65	(1.46, 1.87)‖	1.00	(0.80, 1.25)
COPD	1.73	(1.62, 1.85)‖	1.17	(1.02, 1.33)‡
Air pollutants, $\mu g/m^3$				
PM2.5 yearly average	1.00	(1.00, 1.01)‖	1.00	(1.00, 1.01)‖
PM10 yearly average	1.00	(1.00, 1.01)‖	1.00	(0.99, 1.00)

CI = confidence interval, COPD = chronic obstructive pulmonary disease, PM = particular matter.
* Crude HR, relative hazard ratio.
† Adjusted HR: multivariable analysis including age, sex, and comorbidities of diabetes, hypertension, hyperlipidemia, coronary artery disease, stroke, COPD, and PM2.5 yearly average and PM10 yearly average.
‡ $P < 0.05$.
§ $P < 0.01$.
‖ $P < 0.001$.

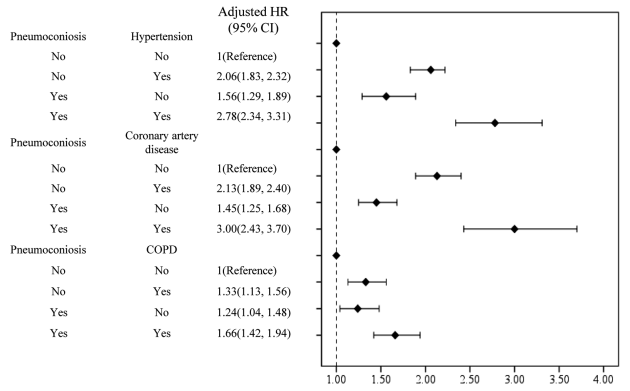

Figure 2. The results of multivariable Cox proportion hazards regression models for the risk of pneumoconiosis and comorbidity contributing to congestive heart failure.

Patients with heart failure in Taiwan have high prevalences of hypertension and diabetes mellitus as their comorbid conditions.[32] The prevalence of heart failure in Taiwan population, we also got the higher and higher incidence (6.06% to 9.71%) of heart failure in Taiwan from 1999 to 2011. In this study, the incidence of CHF in the pneumoconiosis cohort is obviously higher than the general Taiwan population.

The 2 groups were compared after 12 years of follow-up. Compared with the nonpneumoconiosis cohort, the cumulative incidence of CHF was approximately 4.5% higher in the pneumoconiosis cohort, and the overall incidence density of CHF was significantly higher in the pneumoconiosis cohort (crude HR = 1.40). Pneumoconiosis experienced by coal workers is characterized by a progressive fibrotic reaction in the lungs that can cause functional and irreversible damage. Pulmonary fibrosis is an irreversible accumulation of connective tissue in the interstitium of the lungs.[6] A previous study revealed that pneumoconiosis is a risk factor for COPDs such as chronic bronchitis, emphysema, hemoptysis, pneumothorax, pleural disease, infection, autoimmune disease, anthracofibrosis, chronic interstitial pneumonitis, and malignancy.[13] COPD can be implicated by the development of pulmonary hypertension and right heart failure.[26] Cor pulmonale denotes right ventricular hypertrophy secondary to structural or functional abnormalities of the lung.[14] Hypoxemia-induced vasoconstriction and destruction of the pulmonary vascular bed are responsible mechanism.[26] Vasomotor increase in pulmonary vascular resistance as a result of alveolar hypoxia is the most frequent cause of pulmonary heart disease.[14] Complications associated with pneumoconiosis are coughing, breathlessness, and progression to and death from right-sided heart failure.[15]

Increased incidences of specific acute cardiovascular outcomes, including heart failure, have been reported to occur after as little as 1 to 2 hours of increased PM concentration.[27] The emergence evidence and research based on ambient also implicates household air pollution in the development cardiovascular disease.[26] Many epidemiologic studies found positive association between daily variations in air pollution and nonaccidental daily mortality.[28] From the Yang's studies, he also found that short-term adverse effects of air pollution exposure, particularly to CO, NO_2, and O_3, increase hospital admission for CHF in Taiwan.[29,30]

Old age, type 2 diabetes mellitus, and hypertension have been considered risk factors for CHF.[16,17] CAD is the most common cause of CHF in developing countries.[18] CAD is common in heart failure both with reduced ejection fraction and with preserved ejection fraction.[18] In a previous study, apoB/apoA-1 was the strongest risk factor related to the lipoprotein component for developing heart failure.[19] Another study suggested that CHF induces the onset of ischemic stroke in patients with atrial fibrillation.[20]

We observed that the incidence of CHF was higher among the women in both cohorts. However, other studies have indicated differences in CHF incidence between sexes, although the incidence is higher in men, particularly middle-aged men. Heart failure occurs more frequently and develops earlier in men than in women. However, women have a higher risk for developing heart failure with preserved ejection fraction.[21,22] Further study is required to determine the influence of cultural factors. No specific treatment affects the course of CWP, though treatment options are available for complications. Supportive care includes bronchodialtor and pulmonary rehabilitation.[33] To prevent the pneumoconiosis, effective ventilation should be installed,dust should be kept from being spread, and personal protective equipment should be prepared.[25]

In conclusion, our findings suggest that patients with pneumoconiosis are at higher risk of developing CHF than the general population, particularly when the patients also have CAD, hypertension, or COPD.

4.1. Limitation

The strengths of our study include the use of nationwide population-based data that are highly representative of the general population. However, the limitations of these findings must be considered as follows: no available data which may be risk factors for pneumoconiosis or CHF, such as the work environments, daily life activities, socioeconomic conditions, or family histories of related disease in the NHIRD; when comparing with randomized controlled studies, the retrospective cohort study may be lack of possible confounding variables and is lower in evidence and statistical quality; the relevant clinical variables were unavailable in the NHIRD for analyses, such as individual patient's blood pressure, ejection fraction, pneumoconiosis staging, and the classification of CHF.

References

[1] Xia Y, Liu J, Shi T, et al. Prevalence of pneumoconiosis in Hubei, China from 2008 to 2013. Int J Environ Res Public Health 2014;11:8612–21.
[2] Wang T, Yang J, Ji X, et al. Pathway analysis for a genome-wide association study of pneumoconiosis. Toxicol Lett 2014;232:284–92.
[3] Shen F, Yuan J, Sun Z, et al. Risk identification and prediction of coal workers' pneumoconiosis in Kailuan Colliery Group in China: a historical cohort study. PLoS One 2013;8:e82181.
[4] Yucesoy B, Luster MI. Genetic susceptibility in pneumoconiosis. Toxicol Lett 2007;168:249–54.
[5] Lee S, Hayashi H, Maeda M, et al. Environmental factors producing autoimmune dysregulation—chronic activation of T cells caused by silica exposure. Immunobiology 2012;217:743–8.
[6] Lee JS, Shin JH, Lee KM, et al. Serum levels of TGF-β1 and MCP-1 as biomarkers for progressive coal workers' pneumoconiosis in retired coal workers: a three-year follow-up study. Ind Health 2014;52:129–36.
[7] Banumathy S, Dayasagar Rao V, Joshi L, et al. Etiology of congestive heart failure in Indian population—an acute care study of 500 cases. JICC 2013;3:43–8.
[8] Kamath C. A new approach to detect congestive heart failure using sequential spectrum of electrocardiogram signals. Med Eng Phys 2012;34:1503–9.
[9] Marín J, Marín E, Gutiérrez-Iñiguez MA, et al. Mechanisms involved in the hemodynamic alterations in congestive heart failure as a basis for a rational pharmacological treatment. Pharmacol Ther 2000;88:15–31.
[10] Colais P, Faustini A, Stafoggia M, et al. Particulate air pollution and hospital admissions for cardiac diseases in potentially sensitive subgroups. Epidemiology 2012;23:473–81.
[11] Cheng TM. Okma KGH, Crivelli L. Taiwan's National Health Insurance system: high value for the dollar. Six Countries, Six Reform Models: The Health Reform Experience of Israel, the Netherlands, New Zealand, Singapore, Switzerland and Taiwan. Hackensack, NJ:World Scientific; 2009. 71–204.
[12] Kuo HW, Chang CL, Liang WM, et al. Respiratory abnormalities among male foundry workers in central Taiwan. Occup Med (Lond) 1999;49:499–505.
[13] Jun JS, Jung JI, Kim HR, et al. Complications of pneumoconiosis: radiologic overview. Eur J Radiol 2013;82:1819–30.
[14] Murray J, Reid G, Kielkowski D, et al. Cor pulmonale and silicosis: a necropsy based case-control study. Br J Ind Med 1993;50:544–8.
[15] Fishwick D, Barber C. Pneumoconiosis. Medicine 2012;40:310–3.
[16] Verny C. Congestive heart failure in the elderly diabetic. Diabetes Metab 2007;33:S32–9.
[17] Alosco ML, Spitznagel MB, Cohen R, et al. Obesity and cognitive dysfunction in heart failure: the role of hypertension, type 2 diabetes, and physical fitness. Eur J Cardiovasc Nurs 2015;14:334–41.
[18] Lala A, Desai AS. The role of coronary artery disease in heart failure. Heart Failure Clin 2014;10:353–65.

[19] Holme I, Strandberg TE, Faergeman O, et al. Congestive heart failure is associated with lipoprotein components in statin-treated patients with coronary heart disease Insights from the Incremental Decrease in End points Through Aggressive Lipid Lowering Trial (IDEAL). Atherosclerosis 2009;205:522–7.
[20] Shibazaki K, Kimura K, Okada Y, et al. Heart failure may be associated with the onset of ischemic stroke with atrial fibrillation: a brain natriuretic peptide study. J Neurol Sci 2009;281:55–7.
[21] Houde S, Feldman DE, Pilote L, et al. Are there sex-related differences in specialized, multidisciplinary congestive heart failure clinics? Can J Cardiol 2007;23:451–5.
[22] Meyer S, Brouwers FP, Voors AA, et al. Sex differences in new-onset heart failure. Clin Res Cardiol 2015;104:342–50.
[23] Guo YL, Wu T-N, Liou S-H, et al. Occupational medicine in Taiwan. Int Arch Occup Enviorn Health 1999;72:419–28.
[24] Hung YP, Teng CJ, Liu C-J, et al. Cancer risk among patients with coal workers' pneumoconiosis in Taiwan: a nationwide population-based study. Int J Cancer 2014;134:2910–6.
[25] Kuo H-K, Chang C-L, Lai J-S, et al. Prevalence of and factors related to pneumoconiosis among foundry workers in central Taiwan. Science Total Environ 1988;222:133–9.
[26] Bloomfield GS, Lagat DK, Akwanalo OC, et al. Conditions that predispose to pulmonary hypertension and right heart failure in person exposed to household air pollution in LMIC. Global Heart 2012;7: 249–59.
[27] Rich DQ, Freudenberger RS, Ohman-Strickland P, et al. Right hear pressure increases after acute increases in ambient particulate concentration. Environ Health Perspect 2008;116:1167–71.
[28] Yang C-Y. Air pollution and hospital admissions for congestive heart failure in a subtropical city: Taipei, Taiwan. J Toxicol Environ Health A 2008;71:1085–90.
[29] Lee I-M, Tsai S-S, Ho C-K, et al. Air pollution and Hospital admissions for congestive heart failure in a tropical city: Kaohsiung, Taiwan. Inhal Toxicol 2007;19:899–904.
[30] Yang C-Y, Cheng M-H, Chen C-C. Effect of Asia dust storm events on hospital admissions for congestive heart failure in Taipei, Taiwan. J Toxicology Environ Health A 2009;72:324–8.
[31] Chiu C-Z, Cheng J-J. Congestive heart failure in the elderly. Int J Gerontol 2007;1:143–52.
[32] Chen S-M, Chen M-C, Wilson Tang WH. Improving care of patients with heart failure: what can Taiwan accomplish? Acta Cardiol Sin 2007;23:211–24.
[33] Wallaert B. Coal worker's pneumoconiosis. Occup Dis 191–201.
[34] Hsien YL, Tsai SS, Yang CY. Fine particular air pollution and hospital admissions for congestive heart failure: a case-crossover study in Taipei. Inhal Toxicol 2013;25:455–60.