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Tuberculosis increases the subsequent risk of acute coronary syndrome: a nationwide population-based cohort study

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SUMMARY

OBJECTIVE: To evaluate the effects of pulmonary tuberculosis (PTB) on the risk of subsequent acute coronary syndrome (ACS) development.

METHODS: The incidence and risk factors of ACS were investigated in 10 168 newly diagnosed tuberculosis (TB) patients from Taiwan's National Health Insurance Research Database between 1997 and 2010, and 40 672 controls without TB from the general population. The follow-up period ran from the diagnosis of new TB to the date of the ACS event, censoring or 31 December 2010.

RESULTS: During the follow-up period, the overall incidence of ACS was higher in TB patients than in non-TB patients (2.10 vs. 1.51 per 1000 person-years). The incidence of ACS increased by 40% in TB patients after adjusting for age, sex and co-morbidities. Male sex, age, hypertension and diabetes were independent factors for the risk of ACS development. The probability of ACS increased in the years following the TB diagnosis.

CONCLUSION: This nationwide population-based cohort study provides compelling evidence that TB patients are at higher risk of developing ACS, and that the risk increases with age. Clinicians should be aware of this and strive to reduce ACS risk factors in TB patients.

KEY WORDS: TB; ACS; cohort study

ACUTE CORONARY SYNDROME (ACS) represents a group of symptoms attributed to sudden reduced blood flow in the coronary arteries. This syndrome, which includes unstable angina and myocardial infarction with or without ST-segment elevation, is a life-threatening disorder with high morbidity and mortality, despite advances in treatment.[1] Hypertension, diabetes and hyperlipidaemia are well-known cardiovascular risk factors of atherosclerosis development, which contributes to the progression of ACS.[2,3] Cerebrovascular accident (CVA) and cardiovascular diseases (CVD) share similar risks for diseases of the circulatory system. Chronic obstructive pulmonary disease (COPD) associated with reduced lung function is a strong risk factor for cardiovascular events, independent of smoking.[4,5] Iriz et al. showed that direct vessel wall colonisation of *Chlamydia pneumoniae* infection resulted in the progression of atheroma plaques, which is related to ACS,[6] while Roed et al. showed an increased risk of coronary artery disease in patients infected with chronic hepatitis C.[7]

Tuberculosis (TB) remains a serious public health and socio-economic problem. With an incidence of 72.5 cases per 100 000 population, TB remains a common infectious disease in Taiwan.[8] In addition to psychological distress and social stigma, TB also causes disease-related health problems,[9] such as exudative, proliferative and productive inflammation, involving cytokines, chemokines and transcription factors.[10–12] Many researchers have focused on TB prevention and treatment outcome,[9,13,14] and a number of studies have shown that antibodies of mycobacterial heat-shock protein are associated with elevated levels of coronary calcification and early atherosclerosis. This in turn may lead to an increased risk of CVA and CVD through an autoimmune process.[15–17] Sheu et al. showed that TB patients are at an increased risk of ischaemic stroke.[17] However, the association between TB and CVD remains unclear. The present study

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therefore uses epidemiological data to investigate the possibility of increased risk of ACS among TB patients. To the best of our knowledge, this is the first study to address this issue from the perspective of a nationwide population-based cohort.

MATERIALS AND METHODS

Data sources

This nationwide retrospective cohort study was designed to investigate the association between TB and ACS in Taiwan, based on data obtained from the National Health Research Institute, Department of Health. The universal National Health Insurance (NHI) programme, implemented in March 1995, covered approximately 99% of the population of over 23.7 million in Taiwan in 2009.[18] The National Health Insurance Research Database (NHIRD) contains longitudinal claims data for a cohort of 1 million people randomly selected from among all beneficiaries. The NHI RD also provides information on the registry of medical facilities, ambulatory care, details of in-patient orders, dental services, prescription drugs and physicians providing the services. The database ensures confidentiality by scrambling all identifiers according to the data regulations of the NHI, and ensures strengthened data security to protect patient privacy. Several Taiwan studies have demonstrated the high accuracy and validity of diagnosis in the NHIRD.[19,20]

Disease diagnoses were coded according to the International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM). All data were deidentified and analysed anonymously. The study was approved by the Ethics Review Board of China Medical University (CMU-REC-101-012).

STUDY PARTICIPANTS

Exposed cohort

We selected TB patients aged ⩾20 years with TB newly diagnosed from 1997 to 2010 who had received medical care at least three times, including out-patient visits and/or hospitalisations, for a principal diagnosis of TB (ICD-9-CM codes 011-018). The date of TB diagnosis served as the index date. Patients who had a history of ACS before the index date were excluded.

Unexposed cohort

The comparison cohort consisted of randomly selected age-, sex-, and month-matched patients without a diagnosis of TB and/or ACS. The control-to-case ratio was 4:1.

Criteria and definition

Each patient in the study was followed from the index date to a new diagnosis of ACS or until patient cases were censored due to loss to follow-up, death, withdrawal from the insurance system or the end of the follow-up period, on 31 December 2010. ACS diagnosis was based on receipt of medical care at least three times, including out-patient visits and/or hospitalisations, for a principal diagnosis of ACS (ICD-9-CM codes 410 and 411.1). In addition to TB, the following associated co-morbidities with increased risk of ACS development were also included: hypertension (ICD-9-CM codes 401–405), diabetes (ICD-9-CM codes 250), hyperlipidaemia (ICD-9-CM codes 272), CVA (ICD-9-CM codes 430–438), and COPD (ICD-9-CM codes 490–496). Age groups were classified as young adult (⩽40 years), middle-aged adult (41–64 years) and older adult (⩾65 years).

Statistical analysis

All statistical analyses were performed using SAS version 9.2 (SAS Institute Inc, Cary, NC, USA). The Kaplan-Meier survival curve was plotted using R software (R Foundation for Statistical Computing, Vienna, Austria). The distribution of categorical socio-demographic characteristics and baseline co-morbidities was compared between the TB and non-TB cohorts using the χ^2 test. The mean age between both cohorts was measured and tested using the *t*-test. We compared the incidence of ACS between the TB and non-TB cohorts stratified by sex, age and comorbidities. The Poisson regression model was used to assess the incidence rate ratio (IRR) and 95% confidence intervals (CIs) in both cohorts. Multivariate Cox proportional hazard regression models were used to assess the effects of TB on the risk of ACS after adjusting for co-factors significantly related to ACS. Hazard ratios (HRs) and 95%CI were also calculated using the Cox model. Kaplan-Meier analysis and the log-rank test were used to compare the ACS-free incidence for the risk of ACS developing in the TB cohort and the non-TB cohort. All statistical tests were performed at a two-tailed significance level of 0.05.

RESULTS

Demographic characteristics and comorbidities

From the data spanning the years 1997–2010, 10 168 TB and 40 672 non-TB patients were identified. Of these, 68.2% were males. TB incidence increased with age (⩽40 years 17.3%; 41–64 years 36.6%; ⩾65 years 46.2%). The age and sex distribution was similar between the two cohorts. The TB cohort had a significantly higher prevalence of hypertension (38.7% vs. 37.5%), diabetes (21.4% vs. 14.9%), CVA (16.6% vs. 13.7%), and COPD (53.7% vs. 28.5%) than the non-TB cohort (Table 1).

Incidence and hazard ratio of acute coronary syndrome between tuberculosis and non-tuberculosis patients

The overall incidence rate of ACS was 39% higher in TB than in non-TB patients (2.10 vs. 1.51 per

Table 1 Comparison of demographics and co-morbidities between TB patients and controls

	TB		
	No (n = 40 672) n (%)	Yes (n = 10 168) n (%)	P value
Age, years			
⩽40	7 024 (17.3)	1756 (17.3)	0.99
41–64	14 872 (36.6)	3718 (36.6)	
⩾65	18 776 (46.2)	4694 (46.2)	
Age, years, median [IQR]	61.2 [28.2]	62.9 [27.8]	<0.0001*
Sex			
Female	12 924 (31.8)	3231 (31.8)	0.99
Male	27 748 (68.2)	6937 (68.2)	
Co-morbidity			
Hypertension	15 249 (37.5)	3931 (38.7)	0.03
Diabetes	6 072 (14.9)	2180 (21.4)	<0.0001
Hyperlipidaemia	7 126 (17.5)	1714 (16.9)	0.11
CVA	5 583 (13.7)	1692 (16.6)	<0.0001
COPD	11 600 (28.5)	5463 (53.7)	<0.0001

*Mann-Whitney U test.
TB = tuberculosis; IQR = interquartile range; CVA = cerebrovascular disease; COPD = chronic obstructive pulmonary disease.

1000 person-years [py], IRR 1.39, 95%CI 1.30–1.48). After adjusting the covariates, the risk of developing ACS was 1.40-fold higher for TB patients than for the non-TB cohort (adjusted HR [aHR] 1.40, 95%CI 1.14–1.72). Men had a higher incidence of ACS than women in both cohorts (2.37 vs. 1.57/1000 py and 1.73 vs. 1.05/1000 py, respectively). Overall, men had a 41% increase in ACS risk compared with women after adjusting for age and co-morbidities. The incidence of ACS increased with age in both cohorts. The risk of ACS was 6.58-fold higher among middle-aged adults than among young adults (aHR 6.58, 95%CI 3.35–12.90), and 10.20-fold higher among older adults than among young adults (aHR 10.20, 95%CI 5.20–20.20). The incidence of ACS increased in patients with any co-morbidity in both cohorts. TB patients with or without a co-morbidity generally had a higher IRR of ACS development than non-TB patients. Hypertension (aHR 1.94, 95%CI 1.59–2.38) and diabetes (aHR 1.86, 95%CI 1.53–2.27) remained significant factors for an increased risk of ACS development after adjusting for age, sex and other co-morbidities (Table 2).

The Figure shows the Kaplan-Meier curves of the probability of being free of ACS development with number of years after identifying patients in both cohorts. There was a significant difference in ACS occurrence between TB patients and those without TB (log-rank test, P = 0.0014).

DISCUSSION

This study demonstrates a 1.4-fold increased risk in subsequent ACS development among TB patients

Table 2 Comparison of incidence and HR of ACS stratified by sex, age and co-morbidities between TB and non-TB patients

	TB							
		No			Yes		Compared to non-TB	Adjusted
Variable	Cases	py	Rate*	Cases	py	Rate*	IRR (95%CI)	HR (95%CI)†
All	415	274 682	1.51	125	59 623	2.10	1.39 (1.30–1.48)‡	1.40 (1.14–1.72)§
Sex								
Female	94	89 373	1.05	32	20 414	1.57	1.49 (1.32–1.68)‡	1 (Reference)
Male	321	185 308	1.73	93	39 209	2.37	1.37 (1.26–1.49)‡	1.41 (1.15–1.72)‡
Age, years								
⩽40	7	54 049	1.30	2	13 192	1.52	1.17 (0.96–1.42)	1 (Reference)
41–64	123	111 140	11.1	51	24 861	20.5	1.85 (1.67–2.06)‡	6.58 (3.35–12.90)‡
⩾65	285	109 492	26.0	72	21 570	33.4	1.28 (1.16–1.42)‡	10.20 (5.20–20.20)‡
Co-morbidity								
Hypertension								
No	153	185 623	0.82	45	41 166	1.09	1.33 (1.21–1.45)‡	1 (Reference)
Yes	262	89 058	2.94	80	18 457	4.33	1.47 (1.33–1.64)‡	1.94 (1.59–2.38)‡
Diabetes								
No	287	241 012	1.19	73	49 062	1.49	1.25 (1.15–1.35)‡	1 (Reference)
Yes	128	33 669	3.80	52	10 560	4.92	1.30 (1.12–1.49)§	1.86 (1.53–2.27)‡
Hyperlipidaemia								
No	298	234 119	1.27	87	51 060	1.70	1.34 (1.24–1.44)‡	1 (Reference)
Yes	117	40 563	2.88	38	8 563	4.44	1.54 (1.32–1.79)‡	1.18 (0.96–1.45)
CVA								
No	321	245 940	1.31	101	52 613	1.92	1.47 (1.37–1.58)‡	1 (Reference)
Yes	94	28 741	3.27	24	7 010	3.42	1.05 (0.88–1.25)	1.09 (0.88–1.36)
COPD								
No	257	206 273	1.25	45	29 377	1.53	1.23 (1.11–1.36)‡	1 (Reference)
Yes	158	68 408	2.31	80	30 246	2.64	1.15 (1.04–1.27)§	1.07 (0.89–1.28)

*Incidence rate, events per 1000 py.
†Multivariate analysis including age, sex and co-morbidities.
‡$P < 0.001$.
§$P < 0.01$.
HR = hazard ratio; ACS = acute coronary syndrome; TB = tuberculosis; py = person-years; IRR = incidence rate ratio; CI = confidence interval; CVA = cerebrovascular accident; COPD = chronic obstructive pulmonary disease.

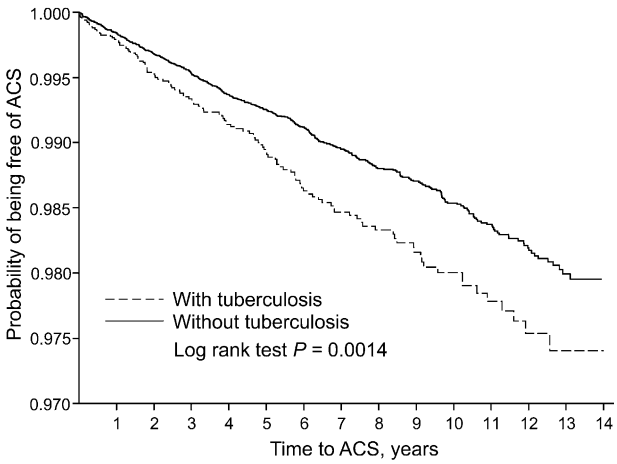

Figure Probability of being free of ACS development for patients with (dashed line) or without (solid line) tuberculosis. ACS = acute coronary syndrome.

compared with controls after adjusting for age, sex and co-morbidities. This is the first study to demonstrate that TB increases the risk of subsequent ACS using epidemiological data. *Mycobacterium tuberculosis* results in chronic granulomatous inflammation, which may be associated with coronary atherosclerosis.[21] Chronic infection is significantly associated with the development of atherosclerosis, the clinical complications of CVD and stroke.[22] Potential mechanisms whereby chronic infections may play a role in atherogenesis are myriad;[22,23] previous studies have shown that the infectious agents with the most evidence of an aetiological role in atherosclerosis include *Chlamydia pneumoniae*, human immunodeficiency virus and viral hepatitis C.[6,7] Smeeth et al. showed a strong association between viral respiratory tract infection and acute myocardial infarction.[24] The increased risk of ischaemic heart disease and stroke related to respiratory tract infections can be greatly attenuated in these patients by influenza vaccination.[25] Van Eeden et al. recently proposed plausible mechanistic pathways through which lung inflammation, systemic inflammatory response and endothelial dysfunction lead to atherosclerosis and plaque disruption that progress to ACS.[23]

In this study most TB patients were men, and the number of cases increased with age. This epiphenomenon is consistent with previous studies.[26,27] Feng et al. showed that male sex was associated with more co-morbidities and poorer treatment outcomes in TB patients in Taiwan.[27] In the present study, men had a higher incidence of ACS than women in both cohorts, and a 41% higher ACS risk than women. This finding is consistent with previous research.[28–30] The overall incidence of ACS increased with age in both cohorts. This may be because older adults have a higher proportion of co-morbidities and an aging process liable to arterial thrombosis in developing ACS.[31] After adjusting for sex and co-morbidities, the risk of ACS still increased with age, which may be associated with the aging process of vessels.[31] Hypertension, diabetes, hyperlipidaemia, CVA and COPD are related to an increased risk of ACS. Hypertension and diabetes are independent factors of ACS risk after adjusting for age, sex and co-morbidities.

The strength of this study is that it provides a retrospective nationwide population-based cohort study to demonstrate the impact of TB on the increased risk of subsequent ACS events. Because each resident in Taiwan is assigned a unique personal identification number, every patient could be traced through the NHI records for the entire follow-up period. However, this study has some limitations. First, the NHIRD does not provide detailed information on cigarette smoking, alcohol consumption, body mass index, physical activity, socio-economic status or family history, all of which are potential confounding factors for this study. Second, all of the participants in both the TB and the non-TB cohorts were patients in the system, which is not representative of the full population, and selection bias could thus be of concern. Third, despite a meticulous study design with adequate control for confounding factors, a key limitation of this study is the potential for bias due to possible unmeasured or unknown confounders.

Although TB is not a traditional risk factor for ACS, this nationwide population-based cohort study shows that TB patients are at greater risk of developing ACS compared with the general population. Increased age, male sex, hypertension and diabetes are also independent risk factors for developing ACS events. In addition to well-known modifiable risk factors, TB is an important risk factor for ACS. To care for TB patients, the government and clinicians should not only focus on a complete treatment, they should also strive to further reduce risk factors for coronary artery disease.

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References

1 Kolansky D M. Acute coronary syndromes: morbidity, mortality, and pharmacoeconomic burden. Am J Manag Care 2009; 15 (Suppl): S36–S41.
2 Picariello C, Lazzeri C, Attana P, Chiostri M, Gensini G F, Valente S. The impact of hypertension on patients with acute coronary syndromes. Int J Hypertens 2011; 2011: 563–657.
3 Vondrakova D, Ostadal P, Kruger A. Immediate effect of intensive atorvastatin therapy on lipid parameters in patients with acute coronary syndrome. Lipids Health Dis 2010; 9: 71.
4 Sin D D, Man S F. Chronic obstructive pulmonary disease as a

risk factor for cardiovascular morbidity and mortality. Proc Am Thorac Soc 2005; 2: 8–11.
5 Huiart L, Ernst P, Suissa S. Cardiovascular morbidity and mortality in COPD. Chest 2005; 128: 2640–2646.
6 Iriz E, Cirak M Y, Engin E D, et al. Effects of atypical pneumonia agents on progression of atherosclerosis and acute coronary syndrome. Acta Cardiol 2007; 62: 593–598.
7 Roed T, Lebech A M, Kjaer A, Weis N. Hepatitis C virus infection and risk of coronary artery disease: a systematic review of the literature. Clin Physiol Funct Imaging 2012; 32: 421–430.
8 Chung W S, Yang M C, Lee M C. Costs and cost effectiveness of directly observed therapy short-course (DOTS) for pulmonary tuberculosis in Taiwan. J Med Health 2012; 1: 33–45.
9 Chung W S, Lan Y L, Yang M C. Psychometric testing of the short version of the World Health Organization quality of life (WHOQOL-BREF) questionnaire among pulmonary tuberculosis patients in Taiwan. BMC Public Health 2012; 12: 630.
10 Sugawara I. Why does tuberculosis lead to specific inflammation? Nihon Hansenbyo Gakkai Zasshi 2009; 78: 263–269.
11 Furuhashi K, Shirai T, Suda T, Chida K. Inflammatory markers in active pulmonary tuberculosis: association with Th1/Th2 and Tc1/Tc2 balance. Kekkaku 2012; 87: 1–7.
12 Bulut Y, Michelsen K S, Hayrapetian L, et al. *Mycobacterium tuberculosis* heat shock proteins use diverse Toll-like receptor pathways to activate pro-inflammatory signals. J Biol Chem 2005; 280: 20961–20967.
13 Chung W-S, Chang Y-C, Yang M-C. Factors influencing the successful treatment of infectious pulmonary tuberculosis. Int J Tuberc Lung Dis 2007; 11: 59–64.
14 Chiang C-Y, Chang C-T, Chang R-E, Li C-T, Huang R-M. Patient and health system delays in the diagnosis and treatment of tuberculosis in Southern Taiwan. Int J Tuberc Lung Dis 2005; 9: 1006–1012.
15 Zhu J, Katz R J, Quyyumi A A, et al. Association of serum antibodies to heat-shock protein 65 with coronary calcification levels: suggestion of pathogen-triggered autoimmunity in early atherosclerosis. Circulation 2004; 109: 36–41.
16 Birnie D H, Holme E R, McKay I C, Hood S, McColl K E, Hillis W S. Association between antibodies to heat shock protein 65 and coronary atherosclerosis. Possible mechanism of action of *Helicobacter pylori* and other bacterial infections in increasing cardiovascular risk. Eur Heart J 1998; 19: 387–394.
17 Sheu J J, Chiou H Y, Kang J H, Chen Y H, Lin H C. Tuberculosis and the risk of ischemic stroke: a 3-year follow-up study. Stroke 2010; 41: 244–249.
18 Cheng T M. Taiwan's National Health Insurance system: high value for the dollar. In: Okma K G H, Crivelli L, eds. Six countries, six reform models: the health reform experience of Israel, the Netherlands, New Zealand, Singapore, Switzerland and Taiwan. New Jersey, USA: World Scientific, 2009: 71–204.
19 Cheng C L, Kao Y H, Lin S J, Lee C H, Lai M L. Validation of the National Health Insurance Research Database with ischemic stroke cases in Taiwan. Pharmacoepidemiol Drug Saf 2011; 20: 236–242.
20 Chung M S, Peng C L, Lin C L, et al. Rheumatoid arthritis increases the risk of deep vein thrombosis and pulmonary thromboembolism: a nationwide cohort study. Ann Rheum Dis 2013; Aug 13. [Epub ahead of print]
21 Rota S. *Mycobacterium tuberculosis* complex in atherosclerosis. Acta Med Okayama 2005; 59: 247–251.
22 Muhlestein J B, Anderson J L. Chronic infection and coronary artery disease. Cardiol Clin 2003; 21: 333–362.
23 Van Eeden S, Leipsic J, Paul Man S F, Sin D D. The relationship between lung inflammation and cardiovascular disease. Am J Respir Crit Care Med 2012; 186: 11–16.
24 Smeeth L, Thomas S L, Hall A J, Hubbard R, Farrington P, Vallance P. Risk of myocardial infarction and stroke after acute infection or vaccination. N Engl J Med 2004; 351: 2611–2618.
25 Nichol K L, Nordin J, Mullooly J, Lask R, Fillbrandt K, Iwane M. Influenza vaccination and reduction in hospitalizations for cardiac disease and stroke among the elderly. N Engl J Med 2003; 348: 1322–1332.
26 Chung W S, Chang R E, Guo H R. Variations of care quality for infectious pulmonary tuberculosis in Taiwan: a population based cohort study. BMC Public Health 2007; 7: 107.
27 Feng J Y, Huang S F, Ting W Y, et al. Gender differences in treatment outcomes of tuberculosis patients in Taiwan: a prospective observational study. Clin Microbiol Infect 2012; 18: E331–E337.
28 Sinha S S, Tremmel J A. Sex differences in acute coronary syndrome. In: SIS yearbook 2007. Seattle, WA, USA: SIS, 2007: 1–8.
29 Emslie C. Women, men and coronary heart disease: a review of the qualitative literature. J Adv Nurs 2005; 51: 382–395.
30 Mujapara A K, Gupta G, Gupta N, et al. Factors affecting coronary arterial disease, comparative study in male vs. female. Int J Pharm Anal 2009; 1: 37–39.
31 De Luca G, van 't Hof A W, Ottervanger J P, et al. Ageing, impaired myocardial perfusion, and mortality in patients with ST-segment elevation myocardial infarction treated by primary angioplasty. Eur Heart J 2005; 26: 662–666.

RÉSUMÉ

OBJECTIF : Evaluer les effets de la tuberculose (TB) pulmonaire sur le risque de l'apparition consécutive d'un syndrome coronaire aigu (ACS).

MÉTHODES : L'incidence et les facteurs de risque d'ACS ont été évalués chez 10 168 patients TB récemment diagnostiqués provenant de la base de données de Recherche de l'Assurance Nationale de Santé de Taiwan entre 1997 et 2010 et 40 672 sujets contrôle non TB de la population générale. La période de suivi s'est étalée du début du nouveau diagnostic de TB à la date de l'événement ACS (élément limitatif) ou jusqu'au 31 décembre 2010.

RÉSULTATS : Au cours de la période de suivi, l'incidence globale de l'ACS a été supérieure chez les patients TB par rapport aux contrôles (2,10 vs. 1,51 pour 1000 années-personne). L'incidence de l'apparition d'ACS augmente de 40% chez les patients TB après ajustement pour l'âge, le sexe masculin et les comorbidités. En outre, le sexe, l'âge, l'hypertension et le diabète constituent des facteurs de risque indépendants de l'apparition d'un ACS. La probabilité d'un ACS augmente au cours des années qui font suite au diagnostic de TB.

CONCLUSION : Cette étude de cohorte basée sur la population et portant sur l'ensemble du pays apporte des preuves irréfutables du fait que les patients TB encourent un risque plus élevé d'apparition d'un ACS et que ce risque augmente avec l'âge. Les cliniciens devraient en être conscients et veiller à réduire les facteurs de risque d'ACS chez les patients TB.

RESUMEN

OBJECTIVO: Evaluar los efectos de la tuberculosis (TB) pulmonar sobre el riesgo de aparición posterior del síndrome coronario agudo (ACS).

MÉTODOS: Se investigó la incidencia del ACS y los factores de riesgo asociados con su aparición en 10 168 pacientes con diagnóstico reciente de TB, a partir de la base de datos del Instituto Nacional de Seguro de Enfermedad de Taiwán entre 1997 y el 2010, y 40 672 testigos sin TB de la población general. El período de seguimiento se extendió desde el establecimiento del diagnóstico de TB hasta el momento del episodio del ACS, la pérdida administrativa (censura) o la fecha del 31 de diciembre del 2010.

RESULTADOS: Durante el período de seguimiento, la incidencia global de ACS fue más alta en los pacientes con TB que en los testigos (2,10 contra 1,51 por 1000 años-persona). La incidencia de aparición del ACS aumentó de 40% en los pacientes con diagnóstico de TB, después del ajuste en función de la edad, el sexo y las enfermedades concomitantes. Además, el sexo masculino, la edad, la hipertensión y la diabetes constituyeron factores independientes de riesgo de aparición del síndrome. La probabilidad de presentación del ACS aumentó en los años posteriores al diagnóstico de TB.

CONCLUSIÓN: El presente estudio de cohortes de escala nacional aporta datos fidedignos convincentes en favor de un mayor riesgo de sufrir el ACS en los pacientes con TB; este riesgo aumenta con la edad. Los médicos deben tener presente esta situación y esforzarse por disminuir los factores de riesgo de aparición del ACS en los pacientes con diagnóstico de TB.