BMJ Open Respiratory Research

Cardiovascular outcomes following hospitalisation for exacerbation of bronchiectasis: a territory-wide study

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To cite: Kwok WC, Tsui CK, Leung SHI, *et al*. Cardiovascular outcomes following hospitalisation for exacerbation of bronchiectasis: a territory-wide study. *BMJ Open Respir Res* 2024;**11**:e001804. doi:10.1136/bmjresp-2023-001804

Received 3 May 2023
Accepted 5 April 2024

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ABSTRACT

Background Although bronchiectasis is reported to be associated with cardiovascular disease, evidence for an association with cardiovascular events (CVEs) is lacking.

Methods A territory-wide retrospective cohort study was conducted in Hong Kong involving all patients who had bronchiectasis diagnosed in public hospitals and clinics between 1 January 1993 and 31 December 2017 were included. Patients were allocated to be exacerbator or non-exacerbator group based on hospitalzied bronchiecsis history and CVEs over the next 5 years determined. Propensity score matching was used to balance baseline characteristics.

Results 10 714 bronchiectasis patients (mean age 69.6±14.4 years, 38.9% men), including 1230 in exacerbator group and 9484 in non-exacerbator group, were analysed. At 5 years, 113 (9.2%) subjects in the exacerbator group and 87 (7.1%) in the non-exacerbator group developed composite CVEs. After adjustment for age, sex, smoking and risk factors for cardiovascular disease, bronchiectasis exacerbation was associated with increased risks for acute myocardial infarction (AMI), congestive heart failure (CHF) and CVE compared with those in the non-exacerbator group with adjusted HR of 1.602 (95% CI 1.006–2.552, p value=0.047), 1.371 (95% CI 1.016–1.851, p value=0.039) and 1.238 (95% CI 1.001–1.532, p=0.049) in the whole cohort. Findings were similar for the propensity score-matched cohort for AMI and CVE.

Conclusion Patients who were hospitalised for exacerbation of bronchiectasis were at significantly increased risk of AMI, CHF and CVE over a 5-year follow-up period.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Although bronchiectasis is reported to be associated with cardiovascular disease, evidence for an association with cardiovascular events is lacking.

WHAT THIS STUDY ADDS

⇒ Patients who were hospitalised for exacerbation of bronchiectasis were at significantly increased risk of acute myocardial infarction, heart failure and composite cardiovascular outcome over a 5-year follow-up period.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Patients with high bronchiectasis exacerbation risk, for example those with high FACED and BSI score, should be closely monitored for any exacerbation and aggressive treatment instigated to prevent recurrent bronchiectasis exacerbations.

INTRODUCTION

Bronchiectasis is a suppurative lung condition characterised by pathological dilatation of the bronchi. The main pathogenesis of bronchiectasis involves airway inflammation, abnormal mucus clearance and bacterial colonisation, resulting in progressive airway destruction and distortion.[1] Typical airway inflammation is neutrophilic with an abundance of neutrophils in sputum, bronchoalveolar lavage fluid and bronchial biopsy, even in patients who are clinically stable.[2]

There is growing evidence of an association of systemic inflammation with cardiovascular diseases.[3] Baseline C reactive protein (CRP) level has been shown to predict the long-term risk of a first myocardial infarction, ischaemic stroke or peripheral artery disease.[4–6] Guidelines suggest that measurement of high-sensitivity CRP in patients at intermediate risk of coronary heart disease (CHD) is appropriate.[7–9] Interleukin 6 (IL-6) and IL-6 receptor have also been suggested to play a direct causal role in the development of CHD. Meta-analyses have confirmed the role of IL-6 and IL-6R in the generation of inflammation and the associated risk of CHD.[10 11] Higher levels of the leucocyte enzyme myeloperoxidase, which is secreted during acute inflammation and promotes oxidation of lipoproteins, are associated with the presence of coronary artery disease and may predict the presence of acute coronary syndrome in patients with chest pain.[9 12–15] Elevated level of other inflammatory markers such as white blood cells, erythrocyte sedimentation rate, IL-18, tumour necrosis

factor-alpha, transforming growth factor-beta, soluble intercellular adhesion molecule-1, P-selectin, cathepsin S and lipoprotein-associated phospholipase A2 have been reported as markers of increased CHD risk.[14 16–22]

As a disease with profound airway inflammation, bronchiectasis has been shown to be associated with the development of cardiovascular disease. Among patients with bronchiectasis, it has been suggested that pulse-wave velocity is significantly higher in frequent exacerbators (≥3 events per year) than infrequent exacerbators (<3 events per year). Frequent exacerbators have been shown to have elevated serum CRP.[23] Patients with bronchiectasis have also been reported to have increased arterial stiffness that correlates with disease severity compared with control subjects.[24] In a population-based study conducted in the UK, a pre-existing diagnosis of CHD (Odds ratios (OR) 1.33, 95% conidence interval (CI) 1.25–1.41) or stroke (OR 1.92, 95% CI 1.85–2.01) was higher in people with bronchiectasis compared with those without, after adjusting for age, sex, smoking and risk factors for cardiovascular disease. The rate of first CHD and stroke was also higher in individuals with bronchiectasis (Hazard ratio (HR) for CHD 1.44 (95% CI 1.27–1.63) and HR for stroke 1.71 (95% CI 1.54–1.90)).[25] In a post hoc analysis of a prospective observational study of 250 patients with bronchiectasis for a median follow-up of 35 months, 74 (29.6%) patients experienced a cardiovascular event (CVE) and 93 (37.2%) died. Age, arterial hypertension, chronic obstructive pulmonary disease and potentially severe exacerbations were factors that significantly increased the risk of developing CVE. Compared with patients without CVE, those with CVE had higher mortality.[26] A recent report suggested that hospitalisation or emergency room visit for bronchiectatic exacerbation was associated with an increase in atherosclerotic cardiovascular disease (ASCaVD)-related or atherosclerotic cerebrovascular disease (ASCeVD)-related mortality within 1 year.[27]

Although there are studies that suggest an association of bronchiectasis and its exacerbation with cardiovascular disease and events, they do have limitations. In a population-based study that suggested the risk of CHD and stroke was higher among patients with bronchiectasis compared with the general population, there was a significant difference in the general population and the bronchiectasis group. Although multivariate analysis was performed, application of propensity score matching would have better addressed the issue of significant differences in the study populations.[25] Other important information about the patients with bronchiectasis, such as *Pseudomonas aeruginosa* colonisation or previous bronchiectasis exacerbation, was also not presented. In another study of the effect of bronchiectasis exacerbation prior to ASCaVD or ASCeVD events on mortality, only 1066 patients were included, despite using a population-based database, and follow-up was for only 1 year. This may have been unable to comprehensively assess the intermediate to long-term effect of bronchiectasis and bronchiectasis exacerbation on CVEs that may take longer to develop.[27] A post hoc analysis of a prospective observational study on the appearance of CVE and mortality had an even smaller sample size although longer follow-up.[26] A dedicated study to assess the effect of bronchiectatic exacerbation and subsequent risk of development of a CVE is warranted.

MATERIALS AND METHODS

This was a territory-wide retrospective cohort study. Data were retrieved on all patients assigned a diagnostic code of bronchiectasis between 1 January 1993 and 31 December 2017. Patients still alive at the study start date of 1 January 2017 from all public hospitals and clinics in Hong Kong were included in the study. Patients with bronchiectasis were identified by International Classification of Diseases, Ninth Revision (ICD-9) code 494 from the Clinical Data Analysis and Reporting System (CDARS) of the Hospital Authority (HA). Baseline characteristics, exposures and outcomes were retrieved. In Hong Kong, the public healthcare service is provided by the HA that manages 43 hospitals and institutions, and 122 outpatient clinics, serving more than 90% of the population. CDARS is an electronic healthcare database managed by the HA, with all essential clinical information including patient demographics, hospitalisation, visits to outpatient clinics and emergency departments, diagnoses, laboratory results, procedures, prescriptions, dispensing of medications and deaths prospectively recorded. This database was established in 1995 for both audit and research purposes. To protect patient confidentiality, each patient is assigned a unique, anonymous patient identifier that is linked to all their clinical data contained in CDARS.

Hospitalisation for bronchiectatic exacerbation was defined as an emergency admission via the Accident and Emergency Department for at least 1 day in 2017 with a principal diagnosis code of bronchiectasis (ICD-9: 494) and oral or intravenous antibiotics prescribed, among all patients with bronchiectasis on active follow-up in the same year. The comparator group was bronchiectatic patients without hospitalisation. The primary outcome was the first subsequent episode of a major CVE, including acute myocardial infarction (AMI), heart failure (CHF), ischaemic stroke (CVA)/transient ischaemic attack (TIA) and intracranial haemorrhage (ICH), and the composite outcome, defined as any of the aforementioned outcomes. Patients lost to follow-up were identified by their last available record from CDARS and censored. The censoring date was set as that of the last entry on CDARS.

Patient and public involvement

Patients and the public were not engaged in any stage during this study.

Statistical analysis

All statistical analyses were performed using R V.4.2.2 (R Foundation for Statistical Computing) statistical software

and 26th version of SPSS statistical package. Continuous variables are expressed as median and IQR. Mann-Whitney U test was used to compare continuous variables of two groups. χ^2 test or Fisher's exact test was applied for categorical variables. Cox regression analysis was used to assess survival. Kaplan-Meier estimator was used for survival function of patients among the two groups. Potential confounders included age, gender, number of cardiovascular risk factors (hypertension, diabetes mellitus, hyperlipidaemia, and atrial fibrillation), history of established cardiovascular and cerebrovascular disease (ischaemic heart disease, ischaemic stroke, TIA and ICH), history of moderate exacerbation and hospitalisation for bronchiectasis exacerbation from 2012 to 2016 and subsequent moderate exacerbation and hospitalisation for bronchiectasis exacerbation in the follow-up period from 2018 to 2022. To better control for the confounders, propensity score adjustment was performed. Patients were matched based on their gender, age, presence of cardiovascular risk factors (hypertension, diabetes mellitus, hyperlipidaemia and atrial fibrillation) and history of established cardiovascular and cerebrovascular diseases (ischaemic heart disease, ischaemic stroke, TIA and ICH), with 1:1 matching and calliper of 0.2 times SD of the logit of propensity score. To further reduce bias from unmeasured confounding, individuals with extreme scores in the upper or lower tail of the propensity score distribution were excluded. Any inadequately matched variables were adjusted in multivariate analysis. Statistical significance was determined at the level of p=0.05.

RESULTS

A total of 10 714 subjects were diagnosed with bronchiectasis during the study period of whom 1230 had a hospitalised exacerbation in 2017 (exacerbator group) and 9484 did not (non-exacerbator group). The patient selection flow chart is illustrated in figure 1. The mean age of the cohort was 69.6±14.4 years and 38.9% were men. The mean follow-up time was 51.9±22.7 months with data cut-off at 31 December 2022. The baseline characteristics of the whole cohort are shown in Tables 1 and 2. Patients in the bronchiectasis exacerbator group were older, more likely to be women, and had higher prevalence of hypertension, diabetes mellitus, hyperlipidaemia, ischaemic heart disease, stroke and TIA, ICH and atrial fibrillation.

Risk of CVEs

Acute myocardial infarction

There were 24 (2.0%) subjects in the exacerbator group and 112 (1.2%) in the non-exacerbator group who developed AMI, with HR 1.708 (95% CI 1.099–2.655, p

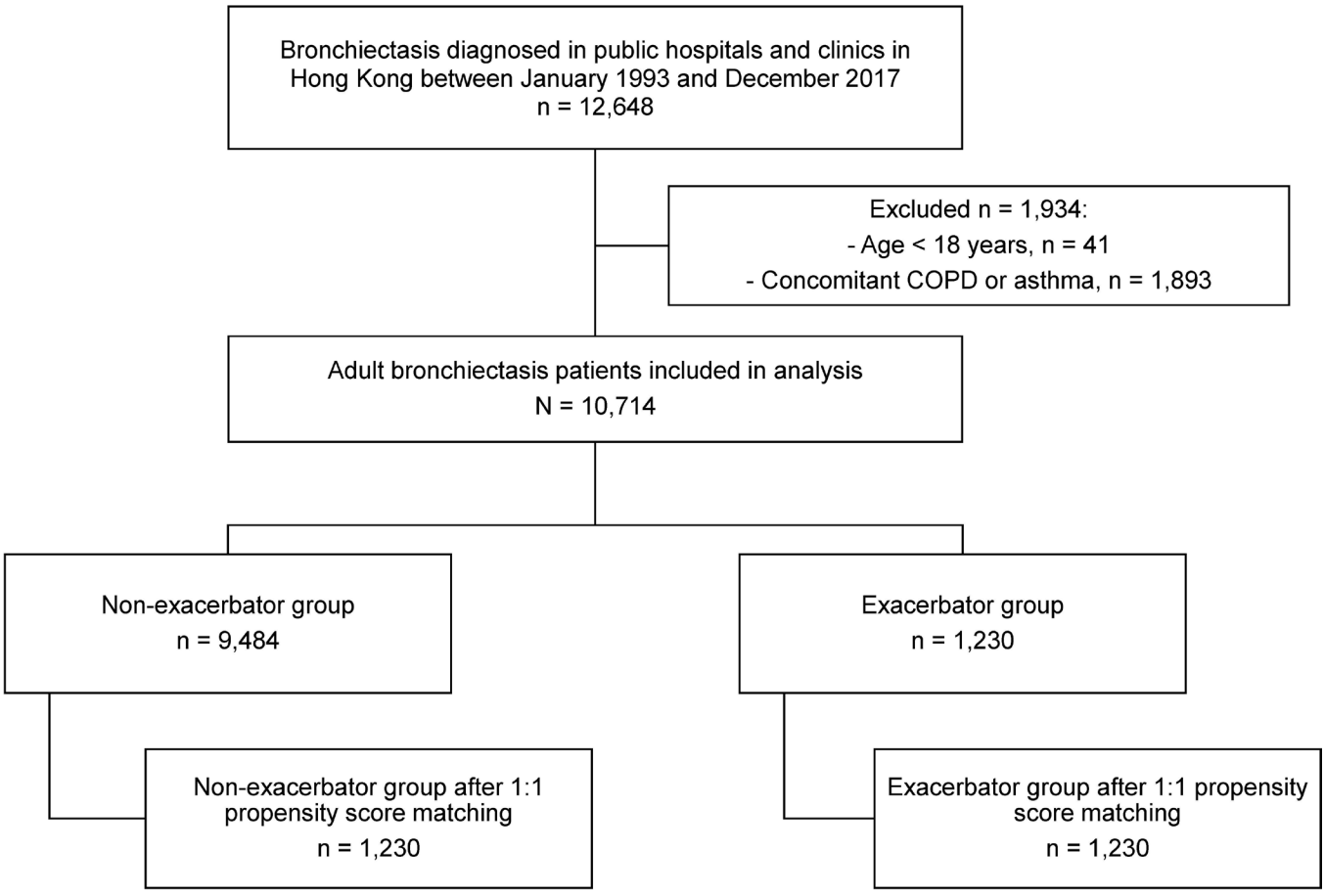

Figure 1 Flow chart of patient selection. COPD, chronic obstructive pulmonary disease.

Table 1 Baseline demographic and clinical characteristics for the whole cohort and propensity score matched cohort

	All	Whole cohort		P value	ASD	PS matched cohort		P value	ASD
	n=10 714	Non-exacerbator (n=9484)	Exacerbator (n=1230)			Non-exacerbator (n=1230)	Exacerbator (n=1230)		
Age, years (mean±SD)	69.60±14.38	69.32±14.48	71.76±13.34	<0.001*	0.176	70.97±13.66	71.76±13.34	0.149	0.058†
Male (N (%))	4163 (38.9)	3722 (39.2)	441 (35.9)	0.024*	0.070†	432 (35.1)	441 (35.9)	0.736	0.015†
Ethnic groups (N (%))				0.275	0.077†			0.236	0.095†
Chinese	10 342 (96.5)	9143 (96.4)	1199 (97.5)			1194 (97.1)	1199 (97.5)		
Other Asian	123 (1.1)	114 (1.2)	9 (0.7)			12 (1.0)	9 (0.7)		
Caucasian	58 (0.5)	55 (0.6)	3 (0.2)			10 (0.8)	3 (0.2)		
Others	191 (1.8)	172 (1.8)	19 (1.5)			14 (1.1)	19 (1.5)		
History of cardiovascular/cerebrovascular events (N (%))									
Ischaemic heart disease	655 (6.1)	540 (5.7)	115 (9.3)	<0.001*	0.139	114 (9.3)	115 (9.3)	1.000	0.003†
Stroke/transient ischaemic attack	344 (3.2)	283 (3.0)	61 (5.0)	<0.001*	0.101	68 (5.5)	61 (5.0)	0.587	0.026†
Intracranial haemorrhage	60 (0.6)	52 (0.5)	8 (0.7)	0.804	0.013†	11 (0.9)	8 (0.7)	0.645	0.028†
Cardiovascular risk factors (N (%))									
Atrial fibrillation	565 (5.3)	472 (5.0)	93 (7.6)	<0.001*	0.107	91 (7.4)	93 (7.6)	0.939	0.006†
Hypertension	1903 (17.8)	1566 (16.5)	337 (27.4)	<0.001*	0.265	330 (26.8)	337 (27.4)	0.786	0.013†
Diabetes mellitus	781 (7.3)	661 (7.0)	120 (9.8)	0.001*	0.101	122 (9.9)	120 (9.8)	0.946	0.005†
Hyperlipidaemia	598 (5.6)	505 (5.3)	93 (7.6)	0.002*	0.091†	111 (9.0)	93 (7.6)	0.214	0.053†

*Statistically significant.
†Good balance with ASD<0.1.
ASD, absolute standardised difference.

value=0.017), suggesting a significantly increased risk in the exacerbator group. The adjusted HR (aHR) was 1.501 (95% CI 1.034–2.180, p value=0.033), after adjustment for age, gender, number of cardiovascular risk factors, history of established cardiovascular disease, number of non-hospitalised bronchiectasis exacerbation between 2012 and 2016, history of hospitalisation for bronchiectasis exacerbation between 2012 and 2016 number of subsequent bronchiectasis exacerbation and hospitalisation for bronchiectasis exacerbation during the follow-up period from 2018 to 2022. The Kaplan-Meier curve is illustrated in figure 2A.

Heart failure

There were 57 (4.6%) subjects in the exacerbator group and 261 (2.8%) in the non-exacerbator group who developed CHF, with HR 1.806 (95% CI 1.355–2.407, p<0.001), suggesting significant increased risk in the exacerbator group. The aHR was 1.280 (95% CI 1.005–1.632, p value=0.046), after adjustment for age, gender, number of cardiovascular risk factors, history of established cardiovascular disease, number of non-hospitalised bronchiectasis exacerbation between 2012 and 2016, history of hospitalisation for bronchiectasis exacerbation between 2012 and 2016, number of subsequent bronchiectasis exacerbation and hospitalisation for bronchiectasis exacerbation during the follow-up period from 2018 to 2022. The Kaplan-Meier curve is illustrated in figure 2B.

Ischaemic stroke and TIA

There were 29 (2.4%) subjects in the exacerbator group and 201 (2.1%) in the non-exacerbator group who developed CVA/TIA. The HR was 1.233 (95% CI 0.834–1.822, p=0.293), suggesting an insignificant risk. The Kaplan-Meier curve is illustrated in figure 2C.

Intracranial haemorrhage

There were 13 (1.1%) subjects in the exacerbator group and 96 (1.0%) in the non-exacerbator group who developed ICH. The HR was 1.665 (95% CI 0.636–1.137, p=0.655), suggesting an insignificant risk. The Kaplan-Meier curve is illustrated in figure 2D.

All cardiovascular/cerebrovascular outcomes

A CVE/cerebrovascular event developed in 113 (9.2%) patients in the exacerbator group and 624 (6.6%) in the non-exacerbator group and included AMI, CHF, CVA/TIA and ICH. The HR was 1.541 (95% CI 1.260–1.884, p<0.001), suggesting significantly increased risk in the exacerbator group. The aHR was 1.205 (95% CI 1.017–1.429, p=0.031) after adjustment for age, gender, number of a risk factors, history of established cardiovascular diseases, history of hospitalisation for bronchiectasis exacerbation in years 2012 to 2016 and subsequent hospitalisation for bronchiectasis exacerbation in the follow-up period from 2018 to 2022. The Kaplan-Meier curve is illustrated in figure 2E.

Propensity score matching

Propensity score matching was performed to match for age, gender, number of cardiovascular risk factors and history of CVEs before the start of follow-up in 2017. After propensity score matching, 1230 subjects in each group were included in the analysis. Subsequent hospitalisation for bronchiectasis exacerbation in the follow-up period from 2018 to 2022 was not well-matched and was adjusted in the multivariate Cox regression model. The mean age of this cohort in 2017 was 71.4±13.5 years and 35.5% were men. The patients were followed up until 31 December 2022. The baseline characteristics of the propensity score-matched subjects are shown in table 1.

Acute myocardial infarction

There were 24 (2.0%) subjects in the exacerbator group and 11 (0.9%) in the non-exacerbator group who developed an AMI, with aHR 2.321 (95% CI 1.111–4.848 p value=0.025) suggesting significant increased risk in the exacerbator group. The Kaplan-Meier curve is illustrated in figure 3A.

Heart failure

There were 57 (4.6%) subjects in the exacerbator group and 42 (3.4%) in the non-exacerbator group who developed CHF. The aHR was 1.170 (95% CI 0.166–1.785, p=0.468), suggesting insignificant risk. The Kaplan-Meier curve is illustrated in figure 3B.

Ischaemic stroke and TIA

There were 29 (2.4%) subjects in the exacerbator group and 24 (2.0%) in the non-exacerbator group who developed ischaemic stroke or TIA (CVA/TIA). The HR was 1.368 (95% CI 0.790–2.368, p=0.264), suggesting insignificant risk. The Kaplan-Meier curve is illustrated in figure 3C.

Intracranial haemorrhage

There were 13 (1.1%) subjects in the exacerbator group and 15 (1.2%) in the non-exacerbator group who developed ICH. The HR was 0.944 (95% CI 0.447–1.996, p=0.880), suggesting insignificant risk. The Kaplan-Meier curve is illustrated in figure 3D.

All cardiovascular/cerebrovascular outcomes

A CVE/cerebrovascular event, which included AMI, CHF, CVA/TIA and ICH, occurred in 113 (9.2%) subjects in the exacerbator group and 87 (7.1%) in the non-exacerbator group. The aHR was 1.359 (95% CI 1.011–1.826, p=0.042), suggesting a significantly increased risk in the exacerbator group. The Kaplan-Meier curve is illustrated in figure 3E.

DISCUSSION

The results of our territory-wide study suggest that severe exacerbation of bronchiectasis that required hospitalisation was a potential risk factor for the occurrence

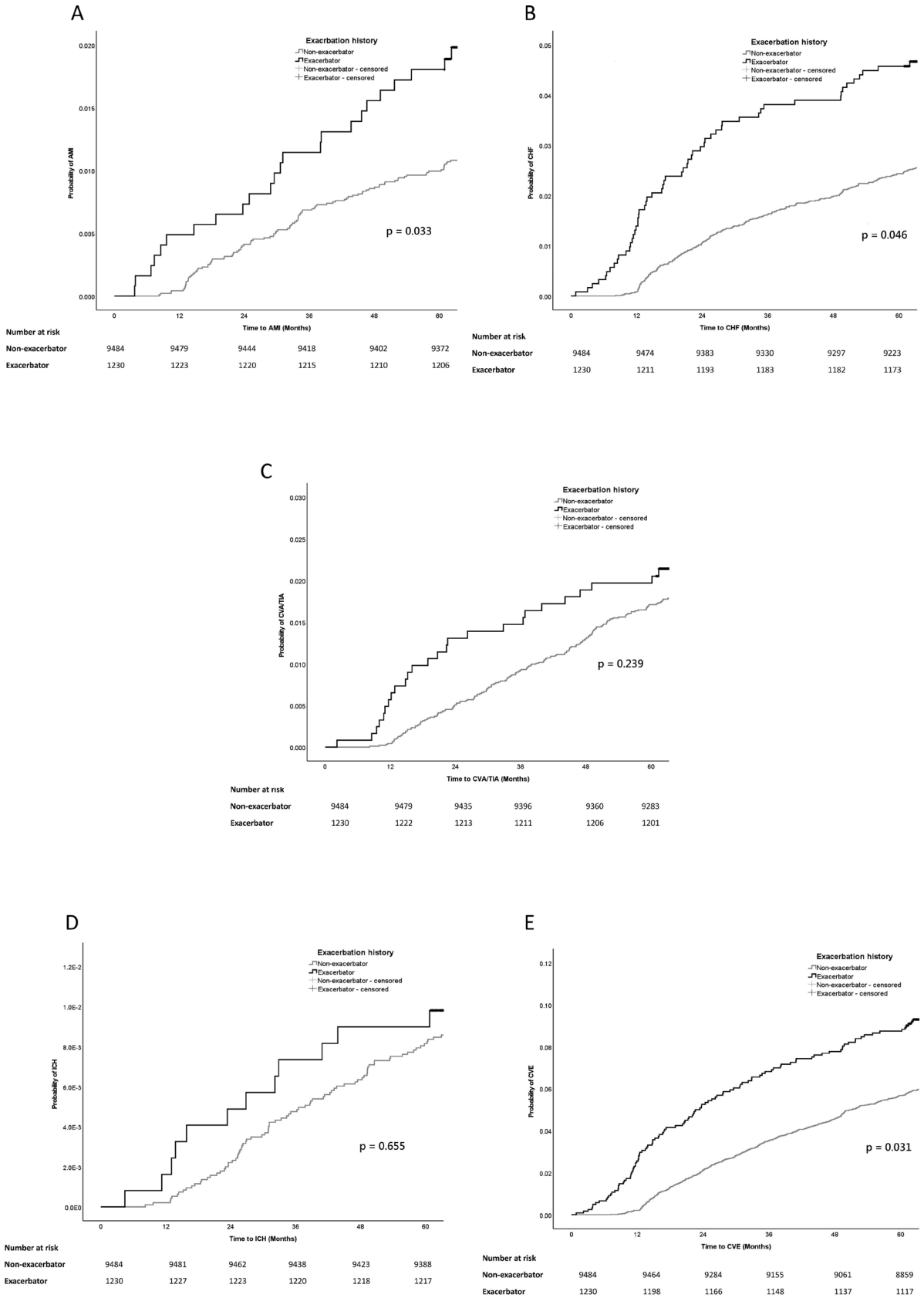

Figure 2 Kaplan-Meier curve for time to events in the whole cohort. (A) Kaplan-Meier curve for time to AMI in the whole cohort. (B) Kaplan-Meier curve for time to CHF in the whole cohort. (C) Kaplan-Meier curve for time to ischaemic stroke or TIA (CVA/TIA) in the whole cohort. (D) Kaplan-Meier curve for time to ICH in the whole cohort. (E) Kaplan-Meier curve for time to CVE in the whole cohort. AMI, acute myocardial infarction; CHF, congestive heart failure; CVE, cardiovascular event; ICH, intracranial haemorrhage; TIA, transient ischaemic attack.

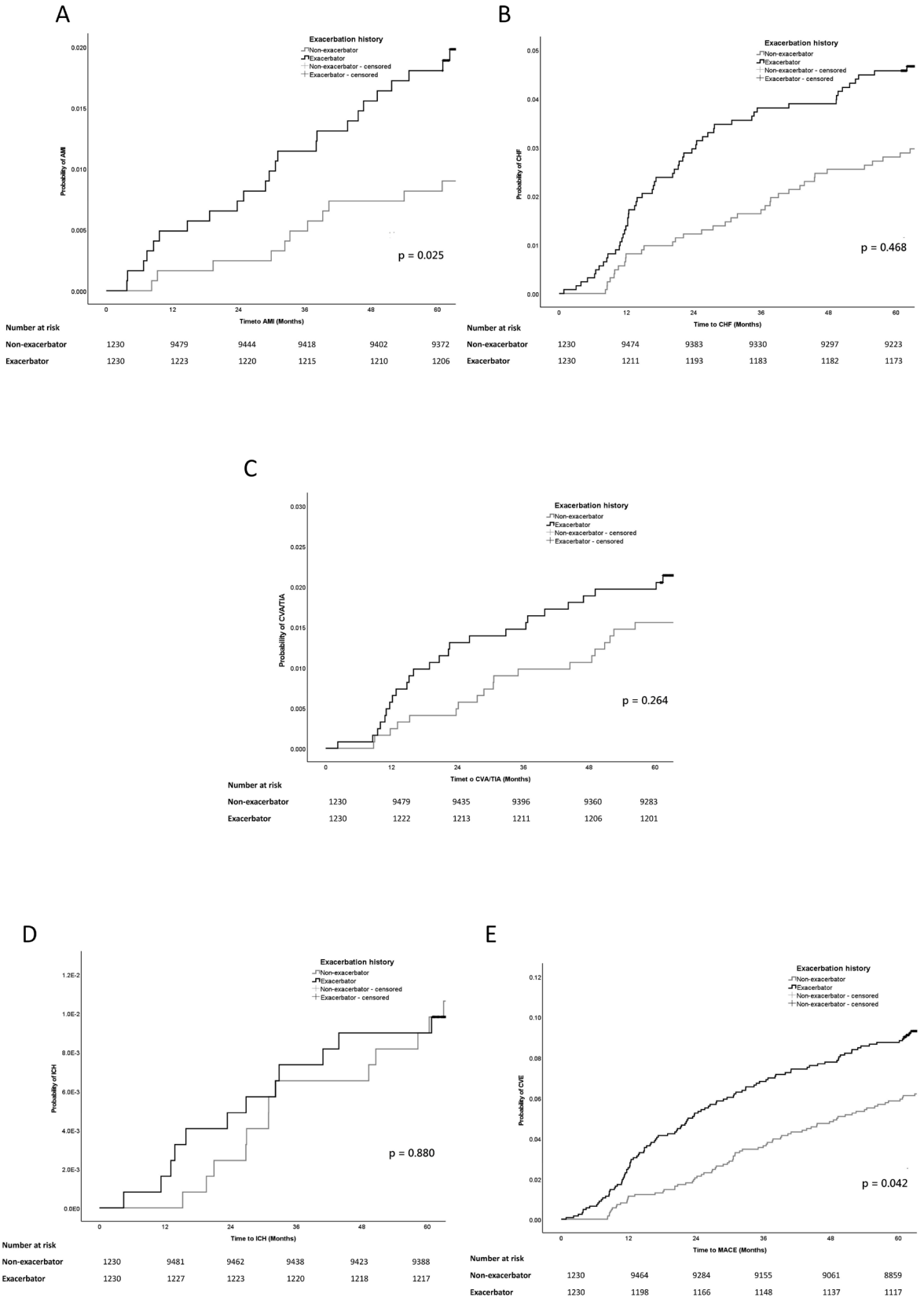

Figure 3 Kaplan-Meier curve for time to events in the propensity score matched cohort. (A) Kaplan-Meier curve for time to AMI in the propensity score matched cohort. (B) Kaplan-Meier curve for time to CHF in the propensity score matched cohort. (C) Kaplan-Meier curve for time to ischaemic stroke or TIA (CVA/TIA) in the propensity score matched cohort. (D) Kaplan-Meier curve for time to ICH in the propensity score matched cohort. (E) Kaplan-Meier curve for time to CVE in the propensity score matched cohort. AMI, acute myocardial infarction; CHF, congestive heart failure; CVE, cardiovascular event; ICH, intracranial haemorrhage; TIA, transient ischaemic attack.

Table 2 Clinical characteristics for the whole cohort and propensity score matched cohort

	All	Whole cohort		P value	ASD	PS matched cohort		P value	ASD
		Non-exacerbator	Exacerbator			Non-exacerbator	Exacerbator		
PsA colonisation (N (%))	1612 (15.0)	1191 (12.6)	421 (34.2)	<0.001*	0.530	421 (34.2)	421 (34.2)	1.000	<0.001†
Laboratory parameters (mean±SD)									
HbA1C, %	5.80±1.33	5.81±1.34	5.73±1.30	0.194	0.060†	5.69±1.56	5.73±1.30	0.666	0.026†
LDL, mmol/L	2.31±0.78	2.31±0.77	2.25±0.80	0.017*	0.087†	2.22±0.76	2.25±0.80	0.535	0.030†
HDL, mmol/L	1.41±0.43	1.41±0.44	1.39±0.41	0.229	0.050†	1.40±0.43	1.39±0.41	0.766	0.016†
Hospitalised bronchiectasis exacerbation (N (%))									
2012–2016	3172 (29.6)	2708 (28.6)	464 (37.7)	<0.001*	0.196	425 (34.6)	464 (37.7)	0.111	0.066†
2018–2022	2047 (19.1)	1484 (15.6)	563 (45.8)	<0.001*	0.691	238 (19.3)	563 (45.8)	<0.001*	0.588
Non-hospitalised bronchiectasis exacerbation (N (%))									
2012–2016	8204 (76.6)	7162 (75.5)	1042 (84.7)	<0.001*	0.232	1010 (82.1)	1042 (84.7)	0.093	0.070†
2018–2022	7318 (68.3)	6264 (66.0)	1054 (85.7)	<0.001*	0.472	899 (73.1)	1054 (85.7)	<0.001*	0.315
≥3 moderate exacerbations per year in 2012–2016	281 (2.6)	207 (2.2)	74 (6.0)	<0.001*	0.194	42 (3.4)	74 (6.0)	0.003*	0.123
≥3 moderate exacerbations per year in 2018–2022	255 (2.4)	192 (2.0)	63 (5.1)	<0.001*	0.167	33 (2.7)	63 (5.1)	0.003*	0.126

*Statistically significant.
†Good balance with ASD<0.1,
ASD, absolute standardised difference; HDL, high-density lipoprotein; LDL, low-density lipoprotein; mmol, millimoles per litre; *PsA*, *Pseudomonas aeruginosa*.

of adverse cardiovascular/cerebrovascular outcomes, including AMI, CVE and probably CHF. The results in our study concur with previous reports of a suggested association of bronchiectatic exacerbation with CVE/cerebrovascular event.

The literature supports a relationship between bronchiectasis and increased risk of cardiovascular disease. The underlying pathogenic mechanism and common risk factors for bronchiectasis and cardiovascular disease are hypoxia and systemic inflammation factors. Recurrent lower respiratory tract infection is also reported to be associated with an acute phase response and increased systemic inflammation, contributing to the increased risk of cardiovascular disease.[27 28] In this study, we identified the increased risk of a CVE following an episode of severe bronchiectasis exacerbation that required hospitalisation. In a small-scale report, CVEs were observed in 29.6% of patients who experienced bronchiectasis exacerbation over a median follow-up of 35 months.[26] Nonetheless the absence of a control group did not enable assessment of the impact of bronchiectasis exacerbation on risk of a CVE.

In contrast, our study compared patients with bronchiectasis and hospitalisation for exacerbation with a similar group who did not require hospitalisation for exacerbation. Our findings suggest that patients who experience an episode of severe bronchiectasis exacerbation will have a significantly increased risk for AMI, CHF and CVEs. The increased risk of CVEs is most reflective of the association between bronchiectasis and CVEs as it includes all cardiovascular/cerebrovascular outcomes. For CHF, the association may reflect more pronounced chronic systemic inflammation in those with exacerbation that led to myocardial strain and consequent CHF. For AMI and CVA/TIA, the association is likely a result of systemic inflammation of bronchiectasis exacerbation. Nonetheless the time required to develop ischaemic heart disease and subsequently CVA/TIA may exceed that of the follow-up duration of this study. A study with prolonged follow-up may better assess this association. Another limitation is that predominantly Chinese patients were included in this study, which might affect the generalisability of the results. Yet, the aetiology of bronchiectasis in Asian especially Chinese population is different from the Caucasians with post-infective bronchiectasis being the most common ones while cystic fibrosis is extremely rare. Having a study with Chinese patients be the dominant ethnic group do have a value to assess the cardiovascular impact in this particular subgroup.

This study suggests that the negative impact of bronchiectasis exacerbation affects not only future exacerbation risks and decline in lung function, but also occurrence of CVEs that may be fatal.[27] The importance of preventing a bronchiectatic exacerbation cannot be overemphasised. Patients with high bronchiectasis exacerbation risk, for example those with high FACED and BSI score, should be closely monitored for any exacerbation and aggressive treatment instigated to prevent recurrent bronchiectasis exacerbations. The benefit of aggressive pharmacological treatment to prevent bronchiectatic exacerbation and consequent cardiovascular outcomes warrants further investigation. Apart from pharmacological treatment, non-pharmacological treatment especially airway clearance physiotherapy to maintain airway hygiene should also be adopted.[29–31] Airway clearance physiotherapy is recommended in European Respiratory Society guidelines.[29 31] Airway clearance physiotherapy has been demonstrated to provide benefits in terms of reduction in the impact of cough, improvement in health-related quality of life (HRQOL) and reduction in the risk of exacerbations.[31] There are different ways in airway clearance including postural drainage, oscillatory positive expiratory pressure, high-frequency chest wall oscillation vest[32] with postural drainage being the most commonly used airway clearance therapy in Hong Kong as it has essentially cost free and with minimal adverse effects. We refer all patients with bronchiectasis to physiotherapist for educating them on how to practice postural drainage. We also checked their compliance to postural drainage on follow-up. Oscillatory positive expiratory pressure and high-frequency chest wall oscillation vest were also offered to selected patients. As airway clearance physiotherapy has been shown to reduce bronchiectasis exacerbation and is a well-recognised non-pharmacological treatment for bronchiectasis, they should be adopted to all patients with bronchiectasis.

CONCLUSION

Hospitalisation for exacerbation of bronchiectasis was associated with a significantly increased risk of AMI, CHF and CVE over a 5-year follow-up.

Acknowledgements The abstract was presented at the 27th Congress of the Asian Pacific Society (APSR) 2023 as poster presentation (33).

Collaborators Nil.

Contributors WCK was involved with the study concept and design, analysis and interpretation of data, acquisition of data, drafting of the manuscript and approval of the final version of the manuscript. CKT, SHIL, CKEW and TCCT were involved with critical revision of the manuscript for important intellectual content and approval of the final version. JCMH was involved with the study concept and design, drafting of the manuscript, critical revision for important intellectual content, study supervision and approval of the final version of the manuscript. JCMH is responsible for the overall content as the guarantor.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and was approved by the Institutional Review Board of the University of Hong Kong and Hospital Authority Hong Kong West Cluster (UW 22-763). Patient consent was waived in this retrospective study by IRB of the University of Hong Kong and Hospital Authority Hong Kong West Cluster as it is a retrospective study without active patient recruitment while the data was already deidentified. The study was conducted with compliance with the Declaration of Helsinki. Patient data was maintained with confidentiality throughout the study.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement All data relevant to the study are included in the article or uploaded as supplementary information.

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