DOI: https://doi.org/10.53555/nnmhs.v8i12.1470 Publication URL: https://nnpub.org/index.php/MHS/article/view/1470

TUBERCULOSIS AND CHRONIC RESPIRATORY DISEASE : A SYSTEMATIC REVIEW

Stephanie talilah[*1]

[*1]*Faculty of Medicine, Indonesian Christian University*

Corresponding Author:-
stephytalilah@yahoo.co.id

Abstract
Tuberculosis, also known as TB, is one of the most common infectious illnesses and has emerged as a major global public health concern. According to statistics provided by the World Health Organization (WHO), there are around 8.6 million new cases of tuberculosis each year, and the disease is responsible for 1.3 million fatalities. More than a half a million cases are documented in youngsters, and an estimated 320,000 fatalities are attributed to those who are infected with HIV. To this day, infectious diseases remain the leading cause of death across the globe, with tuberculosis being the infectious disease that is responsible for the greatest number of deaths overall. This is especially true in nations that are still developing and nations that are emerging. Despite the fact that tuberculosis can show in any organ or tissue, the respiratory system is the site where the illness is most commonly found to be actively spreading. Lungs and airways are both affected by disorders that affect the respiratory system. According to the World Health Organization (WHO), chronic respiratory diseases are responsible for the premature deaths of 4.6 million people each year, which accounts for more than 5% of global mortality. Approximately 90% of these deaths take place in low- and middle-income countries. The findings of this study's search suggested that the prevalence of individuals diagnosed with COPD was noticeably high among patients who had signs of pulmonary TB or who had recovered from pulmonary tuberculosis. There is a dearth of data about other disorders, such as bronchiectasis and bronchitis.

Keyword: *Chronic Respiratory Disease; Inflammation; Pulmonary Disease; Tuberculosis*

INTRODUCTION

Tuberculosis (TB) is one of the main infectious diseases and has become a public health concern worldwide. The World Health Organization (WHO) estimates that there are around 8.6 million incident cases of TB and 1.3 million deaths caused by the disease. More than half a million cases occur in children and 320,000 deaths are reported among people infected with HIV.[1–3] Most of the people who had TB in 2019 were in the WHO Southeast Asia (44%), Africa (25%) and West Pacific (18%) regions, with smaller percentages in the Eastern Mediterranean (8.2%), America (2.9%) and Europe (2.5%). Eight countries accounted for two-thirds of the global total, including: India (26%), Indonesia (8.5%), China (8.4%), Philippines (6.0%), Pakistan (5.7%), Nigeria (4.4%), Bangladesh (3.6%) and South Africa (3.6%).[4]

The two biggest contributors to TB cases are India and Indonesia, where these two countries rank first and second in the world in terms of estimated incident cases per year. N notifications of people newly diagnosed with TB in India increased from 1.2 million to 2.2 million between 2013 and 2019 (+74%), while in Indonesia the number increased from 331,703 in 2015 to 562,049 in 2019 (+69%).[4] The number of new TB cases in Indonesia was 420,994 cases in 2017. The number of new TB cases in 2017 in men was 1.4 times greater than women. Based on the Tuberculosis Prevalence Survey, the prevalence in males is 3 times higher than in females. This may be because men are more exposed to TB risk factors, such as smoking.[5]

Tuberculosis is caused by Mycobacterium tuberculosis, which enters through the respiratory tract. Mycobacterium tuberculosis is an aerobic bacterium that can live mainly in the lungs/various other organs of the body which have high partial pressure. Tuberculosis germs in the form of rods, have special properties that are resistant to acids on staining. TB germs die quickly in direct sunlight, but can survive for several hours in a dark, damp place.[6,7] TB may also cause chronic respiratory diseases like bronchiectasis and COPD. Smoking causes COPD in over 70% of high-income countries and less than 40% of low- and middle-income countries. TB may cause COPD in low- and middle-income countries, but other causes are unknown.[8,9]

Chronic respiratory diseases impact the lungs and airways. Chronic respiratory disease kills 4.6 million people prematurely each year, accounting for more than 5% of worldwide mortality, approximately 90% of which occur in low- and middle-income countries, according to the WHO.[10] COPD was the 9th greatest cause of impairment in the 2010 global burden of illness report and will be the 5th by 2020. COPD, the most common chronic respiratory disease in adults, and bronchiectasis, another debilitating airway disease, have certain clinical symptoms but are commonly overlooked. Airway dilatation and a productive cough cause recurring respiratory infections, lung function decline, and lower quality of life.[11,12]

This article investigate the association between tuberculosis and chronic resporatory disease.

METHODS

Protocol

This systematic review followed the guidelines outlined in the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 checklist, which served as the foundation for the regulations that controlled its conduct..

Eligibility Criteria

This systematic review was developed to analyze papers on "tuberculosis" and "chronic respiratory disease". These are the subjects that were covered in depth in the research that was taken into account. The following requirements have to be met in order for your work to be taken into consideration: 2) Articles have to be written in English. 3) Articles have to have been published after 2012, but before the time this systematic review is created. The following kinds of textual contributions will under no circumstances be considered for inclusion in the anthology: 1) Editorial letters, 2) contributions that do not have a Digital Object Identifier (DOI), and 3) article reviews and submissions that are comparable to those that have previously been published in the journal.

Search Strategy

The search for studies to be included in the systematic review was carried out from November 29th, 2022 using the PubMed and SagePub databases by inputting the words: "tuberculosis" and "chronic respiratory disease". Where *("tuberculosi"[All Fields] OR "tuberculosis"[MeSH Terms] OR "tuberculosis"[All Fields] OR "tuberculoses"[All Fields] OR "tuberculosis"[All Fields]) AND ("chron respir dis"[Journal] OR ("chronic"[All Fields] AND "respiratory"[All Fields] AND "disease"[All Fields]) OR "chronic respiratory disease"[All Fields])* is used as search keywords.

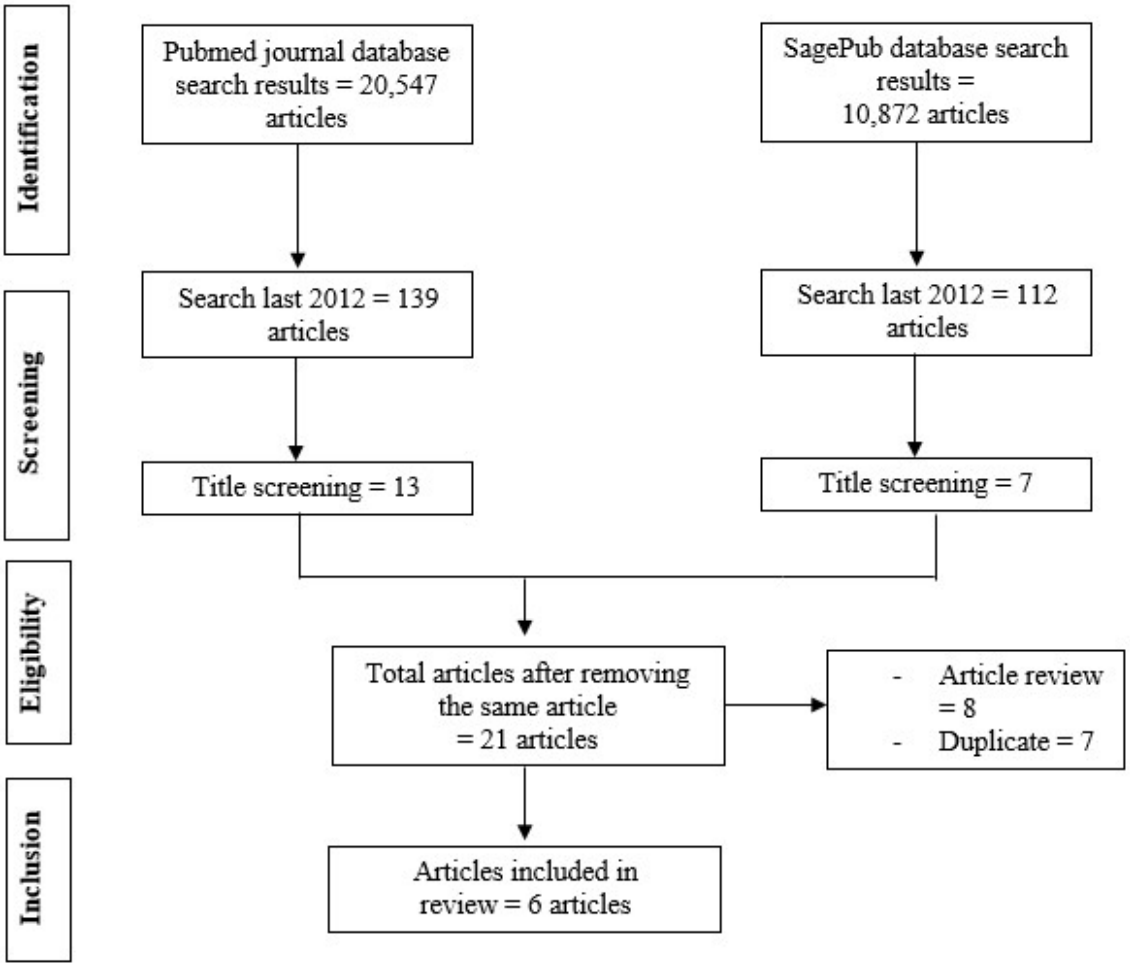

Figure 1. Article search flowchart

Data retrieval

After conducting a literature review and reading the titles and abstracts of previously published studies, the author of the study revised the criteria for what should be included in the study and what should not be included in the study. These changes were made after the author reviewed previously published studies. Only the research projects that were successful in meeting each and every one of the parameters were taken into consideration while the systematic review was being compiled. This was done to ensure that the review is as thorough as possible. It is possible to collect information about each individual study, such as its title, author, publication date, origin of study location, research study design, and research variables. This information can be presented in a number of different formats.

Quality Assessment and Data Synthesis

The writers did their own independent reviews of a selection of the research listed in the titles and abstracts of the papers in order to decide which studies could be appropriate for consideration. After this step, the full texts of the studies that meet the criteria for inclusion in the systematic review will be read in order to determine which studies can be utilized as final inclusions for the purpose of the review. This will be done so in order to answer the question, "Which studies can we use for the purpose of the review?"

RESULT

After 75.8–65.4 days had passed since the beginning of the research, anti-TB medication was first administered. Diabetes mellitus was a protective factor against COPD, but age, being male, having a poor income, and having pulmonary tuberculosis were risk factors (hazard ratio = 2.054 [1.768–2.387]). After a diagnosis, tuberculosis impacted women and people over the age of 70 for a period of six years. Patients with tuberculosis who delayed receiving anti-TB treatment showed a dose-response connection with their chance of developing COPD.[13]

Table 1. The litelature include in this study

Author	Origin	Method	Sample Size / Characteristic	Result
Lee, 2012[13]	Taiwan	Retrospective study	3,176 pulmonary TB cases and 15,880 control subjects	Anti-TB medication started 75.8±65.4 days after the research began. Diabetes mellitus protected against COPD, while age, male, low income, and pulmonary TB were risk factors (hazard ratio = 2.054 [1.768–2.387]). TB affected women and people over 70 for six years following diagnosis. TB patients who delayed anti-TB treatment demonstrated a dose-response connection with COPD risk.
Zhou, 2013[14]	China	Cross sectional	10,811 patients	Respiratory infection during childhood (suffering two kinds of respiratory diseases vs never; OR = 4.89, 95% CI 2.03-11.81), exposure to coal (OR = 2.30, 95% CI 1.17-4.52), chronic pharyngitis (OR = 3.96, 95% CI 1.38-11.40) and pulmonary tuberculosis (OR = 3.07, 95% CI 1.89-4.98), heart diseases (OR = 1.64, 95% CI 1.11-2.42) and lung cancer(OR = 18.61, 95% CI 7.67-45.18).
Amaral, 2015[15]	UK	Cross sectional	10,811 patients	Self-reported tuberculosis was linked with airflow obstruction (adjusted odds ratio [aOR] = 2.51, 95% CI = 1.83-3.42) and spirometric limitation (adjusted odds ratio [aOR] = 2.13, 95% CI = 1.42-3.19). In areas where tuberculosis is common, airflow obstruction and spirometric limitation may be a cause of obstructive illness and impaired lung function.
Choi, 2017[16]	Republic of Korea	Cross sectional	13,522 subjects	Airflow obstruction (AFO) was linked with only a history of TB (OR = 1.53, 95% CI = 0.95-2.46), only TB lesions on CXR (OR = 2.37, 95% CI = 1.80-3.12), and both (OR = 4.47, 95% CI = 3.07-6.51) after adjustment for gender, age, body mass index, education, income, and smoking quantity (P for trend < 0.001). After correction for the aforesaid variables, spirometric limitation was linked with simply a history of TB, only TB lesions on CXR, or both (OR = 2.65, 95% CI = 1.74-4.05) (P for trend < 0.001).
Dutta, 2015[17]	India	Cross sectional	35,948 patients	Women had 2.7% chronic bronchitis. Chronic bronchitis was linked to older age, a cattle shed on the property, fertilizer storage, allergy history, and pulmonary tuberculosis.
Echazarreta, 2018[18]	Argentina	Cross sectional	4,599 surveys	COPD was found to have a significant association with age (odds ratio of 3.77 in individuals 50-59 years of age and 19.23 in those > 80 years), male gender (odds ratio of 1.62; confidence interval [CI]: 1.31-2), smoking (odds ratio of 1.95; CI: 1.49-2.54), low socioeconomic status (odds ratio of 1.33; CI: 1.02-1.73), and previous tuberculosis (OR 3.3; CI 1.43-7.62).

Prevalence of bronchiectasis increased with age ($\chi^2 = 31.029$, $P < 0.001$). There were no statistical significances in crude prevalences of bronchiectasis among cities ($\chi^2 = 10.572$, $P = 0.103$), while there was a significant difference among cities after adjustment with confounders (Wald value = 22.116, P = 0.001), by using logistic regression analysis. Logistic regression analysis showed, bronchiectasis was significantly associated with elder (≥ 70 years vs 40-49 years; OR = 4.11, 95% CI 2.29-7.36), the family history of respiratory diseases (having two subjects with respiratory diseases in family vs no suffered relatives; OR = 2.04, 95% CI 1.06-3.94), respiratory infection during childhood (suffering two kinds of respiratory diseases vs never; OR = 4.89, 95% CI 2.03-11.81), exposure to coal (OR = 2.30, 95% CI 1.17-4.52), chronic pharyngitis (OR = 3.96, 95% CI 1.38-11.40) and pulmonary tuberculosis (OR = 3.07, 95% CI 1.89-4.98), heart diseases (OR = 1.64, 95% CI 1.11-2.42) and lung cancer(OR = 18.61, 95% CI 7.67-45.18).[14]

Airflow obstruction (adjusted odds ratio 2.51, 95% confidence interval [CI] 1.83-3.42) and spirometric limitation (adjusted odds ratio 2.13, 95% confidence interval [CI] 1.42-3.19) were found to be linked with a self-reported history of tuberculosis. A previous diagnosis of tuberculosis was found to be associated with airflow obstruction as well as spirometric restriction. This finding suggests that tuberculosis should be considered as a potentially important cause of obstructive disease and low lung function, particularly in areas where tuberculosis is prevalent.[15]

After adjustment for gender, age, body mass index, education, income, and smoking amount (P for trend 0.001), airflow obstruction (AFO) was associated with only a history of TB (OR = 1.53, 95% CI = 0.95-2.46), only TB lesion on CXR (OR = 2.37, 95% CI = 1.80-3.12), and both a history and TB lesions on CXR (OR = 4.47), spirometric limitation was linked with only a history of tuberculosis (OR = 1.29, 95% CI = 0.80-2.08), only TB lesions on CXR (OR = 2.03, 95% CI = 1.49-2.76), and both a history and TB lesions on CXR (OR = 2.65, 95% CI = 1.74-4.05) (P for trend 0.001). It is possible that the relationship between tuberculosis and respiratory dysfunction may be underestimated if TB lesions will not be considered on CXR.[16]

Dutta's study in India was shown that 2.7% of women suffer from chronic bronchitis on a regular basis. Significant correlates of chronic bronchitis have been identified, including factors such as older age, the existence of a cattle stable

within the house's premises, the storage of fertilizers inside the house, a history of allergies, and a history of lung tuberculosis.[17] COPD was found to have a significant association with age (OR = 3.77 in individuals 50-59 years of age and 19.23 in those > 80 years), male gender (OR = 1.62; 95% CI = 1.31-2), smoking (OR = 1.95; 95% CI = 1.49-2.54), low socioeconomic status (OR = 1.33; CI: 1.02-1.73), and previous tuberculosis (OR = 3.3; 95% CI = 1.43-7.62).[18]

DISCUSSION

Infectious diseases continue to be the greatest cause of mortality worldwide, and tuberculosis is the infectious disease that causes the most deaths overall. This is especially true in less developed and emerging nations. The respiratory system is the most prevalent site of active tuberculosis, despite the fact that the disease can manifest in any organ or tissue. Without therapy, tuberculosis causes the chronic inflammation that is characteristic of the disease and has a mortality rate of fifty percent within five years.[19]

Non-adherence to anti-TB medication, despite the fact that standard treatment for tuberculosis is very effective, with a rapid remission of symptoms and a low rate of relapse, continues to be a major barrier to successful treatment. About two-thirds of individuals, even after completing treatment for pulmonary tuberculosis, continue to have abnormalities in their pulmonary function, with obstructive defect being the most significant anomaly.[19–21]

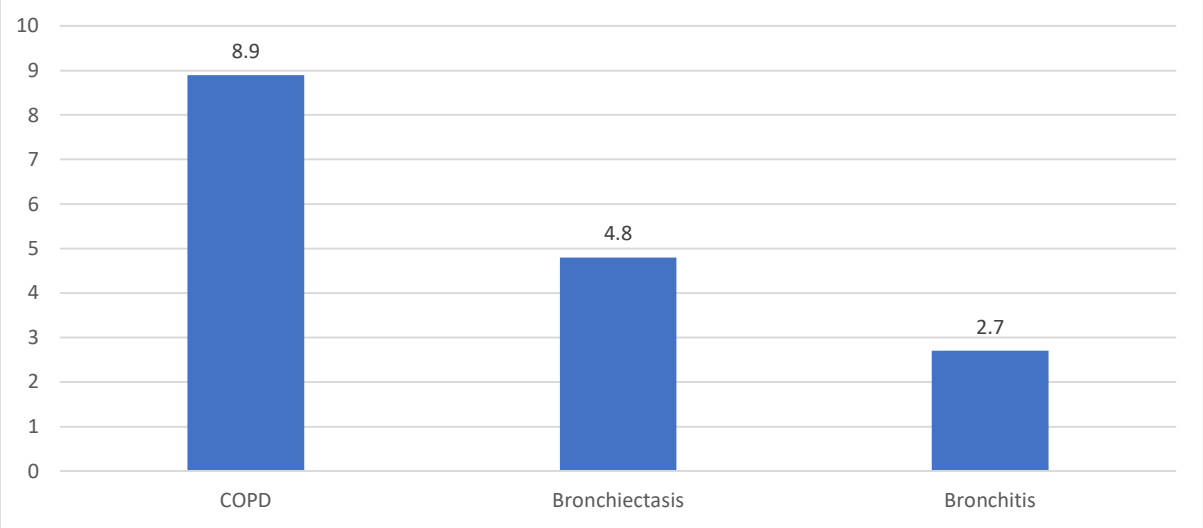

Figure 2. The incidence rate of chronic pulmonary disease after or concomitant with TB

If treatment is not sought for tuberculosis, the disease will lead to chronic caseous granulomatous inflammation, which will, in turn, result in catastrophic damage to the affected tissues. Pulmonary tuberculosis is the most common site of involvement, and it has the potential to cause permanent pulmonary function impairment, either obstructive or restrictive. This, in turn, has the potential to contribute to the pathogenesis of COPD.[8,12]

The earliest manifestations of tuberculosis are notoriously vague and non-specific, making it simple to confuse them with symptoms of other more prevalent clinical conditions. It is important to start therapy as soon as possible for individuals with pulmonary tuberculosis, regardless of whether they have a positive or negative smear test. Delays in identification and treatment may have long-term pulmonary repercussions and may make the development of COPD more likely. This is in addition to making TB more severe and increasing the likelihood that it will be passed on to others.[19,20]

There are a number of additional possibilities that could account for variations between research. First is the foundation that serves as the basis for the designation, previous PTB. "Self-reporting" by subjects was the method that was employed in the majority of the examined research, despite the fact that the questions that were asked were not documented in the vast majority of the cases. A study of hospital or clinic files for a recorded history of PTB or treatment delivered for PTB, CXR findings compatible with previous PTB, and even bacteriologically proved PTB in the past are some other procedures that can be used.[22]

Each technique possesses a unique set of performance characteristics and may deliver varying degrees of associational power. Self-reporting of a previous episode of bacteriologically confirmed PTB treated for 4 or more months (or similar) may be sufficient for the purposes of epidemiology. Self-reporting is subject to patient characteristics, such as underreporting due to stigmatization of the condition, which might be a problem.[22]

Even with the deployment of a liquid mycobacterial culture technique, the diagnosis of pulmonary tuberculosis in patients with a smear-negative illness still takes at least two weeks. Both COPD and tuberculosis require a high index of clinical suspicion and early mycobacteriologic tests. These studies can include acid-fast smears, mycobacterial culture, and even a rapid molecular diagnostic test.[19,20]The evidence that tuberculosis is a risk factor for the future development of bronchiectasis is less substantial than the evidence that it is a risk factor for chronic obstructive pulmonary disease (COPD). Only two studies, both from eastern Asia, were taken into consideration for this article. The findings of the study showed favorable relationships that were similar and substantial. A history of pulmonary tuberculosis was found to be substantially linked with the medical diagnosis of bronchiectasis (OR 3.07 with 95% CI 1.89-4.98) in a large population-based cross-sectional investigation of persons aged over 40 years residing in urban China (n = 10,811).[14]

It is still not completely understood how pulmonary tuberculosis might progress to COPD. Previous research has suggested that a relationship may exist between COPD and rapid emphysematous change, which is produced by residual chronic or recurring inflammation as a result of TB, and dysregulation of macrophages, which is caused by latent intracellular infection.[23,24] It is common knowledge that macrophages play a central role in the remodeling that leads to chronic airflow obstruction. Although the primary function of macrophages in the lung is to kill bacteria or to promote wound healing and resolution, it is also common knowledge that macrophages contribute to the remodeling that leads to chronic airflow obstruction.[24]

There is a possibility that mycobacteria could be present in lung macrophages, which could lead to the continuation of lung inflammation and more severe airway alterations. Other mechanisms include the loss of parenchymal tissue and scarring that occurs after TB, as well as structural damage to the air passages and small airways, such as bronchiectasis as a result of endobronchial involvement, bronchial stenosis, and bronchiolitis occlusion as a result of peribronchial fibrosis.[25]

Other mechanisms include: According to the findings of another study, tuberculosis can increase the activity of matrix metalloproteinases, which can result in aberrant breakdown of the extracellular matrix of the lung. As a result, lung function can be reduced. In order to help inform prognostic and therapeutic options for post-TB lung disease, it will be necessary for future research to understand the underlying immunological processes that contribute to TB-associated COPD in relation to the natural history of TB disease.[26]

CONCLUSION

The search results in this study indicated that the prevalence of patients with COPD was clearly high in post-pulmonary TB patients or patients with evidence of pulmonary TB. Data for other conditions such as bronchiectasis and bronchitis are lacking.

REFERENCE

[1].World Health Organization (WHO). WHO global tuberculosis report 2016. Geneva; 2017.
[2].WHO. Global Tuberculosis Report 2019. 2019.
[3].Schwartz N, Price S, Pratt R. Tuberculosis - United States. Morb Mortal Wkly Rep. 2020;69(11):286–9.
[4].WHO.Global tuberculosis report 2021 [Internet]. World Health Organization. 2021. Tersedia pada: https://www.who.int/publications/digital/global-tuberculosis-report-2021
[5].Kementerian Kesehatan Republik Indonesia. Infodatin. Jakarta; 2018.
[6].Setiati S, Alwi I, Sudoyo AW, Sumadibrata M, Setiyohadi B, Syam AF. Buku Ajar Ilmu Penyakit Dalam. 6 ed. Jakarta: Interna Publishing; 2014.
[7].Fishman JA, Grippi MA, Kotloff RM, et al. Fishman's Pulmonary Disease and Disorders Fifth Edition. New York: Elsevier Saunder; 2016.
[8].Buist AS, Vollmer WM, McBurnie MA. Worldwide burden of COPD in high- and low-income countries. Part I. The burden of obstructive lung disease (BOLD) initiative. Int J Tuberc lung Dis Off J Int Union against Tuberc Lung Dis. Juli 2008;12(7):703–8.
[9].Lam KH, Jiang CQ, Jordan RE, Miller MR, Zhang W Sen, Cheng KK, et al. Prior TB, smoking, and airflow obstruction: a cross-sectional analysis of the Guangzhou Biobank Cohort Study. Chest. Maret 2010;137(3):593–600.
[10]. Hnizdo E, Singh T, Churchyard G. Chronic pulmonary function impairment caused by initial and recurrent pulmonary tuberculosis following treatment. Thorax. Januari 2000;55(1):32–8.
[11]. Labaki WW, Rosenberg SR. Chronic Obstructive Pulmonary Disease. Ann Intern Med. Agustus 2020;173(3):ITC17–32.
[12]. Fishman JA, Grippi MA, Kotloff RM, et al, Fishman JA, Grippi MA, Kotloff RM et al, Fishman JA, et al. Fishman's Pulmonary Disease and Disorders Fifth Edition. New York: Elsevier Saunder; 2016.
[13]. Lee C-H, Lee M-C, Lin H-H, Shu C-C, Wang J-Y, Lee L-N, et al. Pulmonary Tuberculosis and Delay in Anti-Tuberculous Treatment Are Important Risk Factors for Chronic Obstructive Pulmonary Disease. PLoS One [Internet]. 25 Mei 2012;7(5):e37978. Tersedia pada: https://doi.org/10.1371/journal.pone.0037978
[14]. Zhou Y, Wang C, Yao W, Chen P, Kang J, Huang S, et al. [The prevalence and risk factors of bronchiectasis in residents aged 40 years old and above in seven cities in China]. Zhonghua nei ke za zhi [Internet]. 2013;52(5):379–82. Tersedia pada: http://europepmc.org/abstract/MED/23945301
[15]. Amaral AFS, Coton S, Kato B, Tan WC, Studnicka M, Janson C, et al. Tuberculosis associates with both airflow obstruction and low lung function: BOLD results. Eur Respir J. Oktober 2015;46(4):1104–12.
[16]. Choi CJ, Choi WS, Lee SY, Kim KS. The Definition of Past Tuberculosis Affects the Magnitude of Association between Pulmonary Tuberculosis and Respiratory Dysfunction: Korea National Health and Nutrition Examination Survey, 2008-2012. J Korean Med Sci. Mei 2017;32(5):789–95.
[17]. Dutta S, Deshmukh PR. Prevalence and determinants of self-reported chronic bronchitis among women in rural Central India. Med journal, Armed Forces India. Januari 2015;71(1):48–52.
[18]. Echazarreta AL, Arias SJ, Del Olmo R, Giugno ER, Colodenco FD, Arce SC, et al. Prevalence of COPD in 6 Urban Clusters in Argentina: The EPOC.AR Study. Arch Bronconeumol. Mei 2018;54(5):260–9.
[19]. Jain NK. Chronic obstructive pulmonary disease and tuberculosis. Lung India. 2017;34(5):468–9.
[20]. Hooper R, Burney P, Vollmer WM, McBurnie MA, Gislason T, Tan WC, et al. Risk factors for COPD spirometrically defined from the lower limit of normal in the BOLD project. Eur Respir J. Juni 2012;39(6):1343–53.

[21]. Jain NK, Thakkar MS, Jain N, Rohan KA, Sharma M. Chronic obstructive pulmonary disease: Does gender really matter? Lung India. Oktober 2011;28(4):258–62.
[22]. Allwood BW, Myer L, Bateman ED. A systematic review of the association between pulmonary tuberculosis and the development of chronic airflow obstruction in adults. Respiration. 2013;86(1):76–85.
[23]. Chakrabarti B, Calverley PMA, Davies PDO. Tuberculosis and its incidence, special nature, and relationship with
[24]. Holloway RA, Donnelly LE. Immunopathogenesis of chronic obstructive pulmonary disease. Curr Opin Pulm Med. Maret 2013;19(2):95–102.
[25]. Mosser DM, Edwards JP. Exploring the full spectrum of macrophage activation. Nat Rev Immunol. Desember 2008;8(12):958–69.
[26]. Elkington PTG, Friedland JS. Matrix metalloproteinases in destructive pulmonary pathology. Thorax. Maret 2006;61(3):259–66.