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RESEARCH ARTICLE

PREVIOUS PULMONARY TUBERCULOSIS AND COPD OUTCOMES

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Abstract

Background: -Pulmonary tuberculosis (TB) is a recognized cause of permanent structural lung damage and post-tuberculosis lung disease (PTLD), both of which may contribute to the development and progression of chronic obstructive pulmonary disease (COPD). Although previous TB has been associated with impaired lung function, its impact on COPD outcomes remains incompletely understood. This study aimed to evaluate the influence of previous pulmonary TB on exacerbation burden and mortality among patients with COPD.

Methods:-We conducted an observational cohort study involving consecutive patients with spirometry-confirmed COPD managed at the Pulmonology Department of Hassan II Hospital, Agadir, Morocco, between January 2022 and March 2024. Demographic characteristics, smoking history, clinical features, COPD severity, treatment, exacerbation frequency, and previous pulmonary TB were recorded at enrollment. Patients were followed until March 2025 to assess survival. Kaplan–Meier analysis was used to compare survival between groups, and Cox proportional hazards regression was performed to identify independent predictors of mortality.

Results:-A total of 157 patients were included, including 65 (41.4%) with a history of pulmonary tuberculosis. Patients with previous TB experienced significantly more frequent COPD exacerbations than those without previous TB (p = 0.017), despite a lower prevalence of current smoking (21.7% vs. 36.3%, p = 0.004). No significant differences were observed in hospitalization rate, GOLD classification, CAT score,chronic respiratory failure, or in-hospital mortality. Kaplan–Meier analysis demonstrated a significant difference in survival between the two groups (log-rank p = 0.036). However, after adjustment for potential confounders, previous TB was not independently associated with mortality (HR 1.63, 95% CI 0.56–4.77; p = 0.369), whereas the annual exacerbation rate remained the only independent predictor of mortality (p < 0.001).

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Conclusions:-Previous pulmonary tuberculosis was associated with a greater exacerbation burden but not with an increased risk of mortality after multivariable adjustment in patients with COPD. These findings reinforce the concept of post-tuberculosis lung disease as a distinct clinical phenotype of COPD and underscore the importance of early recognition and optimized long-term management to reduce exacerbations and improve patient outcomes.

Introduction:-

Chronic obstructive pulmonary disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation and chronic airway inflammation. It remains one of the leading causes of morbidity and mortality worldwide and imposes a substantial healthcare and socioeconomic burden. Although cigarette smoking is the principal risk factor, COPD is increasingly recognized as a heterogeneous disease resulting from the interaction of multiple environmental and host-related factors, including biomass smoke exposure, air pollution, genetic susceptibility, and previous pulmonary infections(1,2).

Pulmonary tuberculosis (TB) continues to represent a major public health challenge, particularly in low- and middle-income countries where its long-term respiratory consequences are increasingly recognized. Despite successful microbiological treatment, many patients develop permanent structural and functional pulmonary abnormalities, a condition now referred to as post-tuberculosis lung disease (PTLD). Residual fibrosis, bronchiectasis, emphysematous changes, pleural thickening, and chronic airflow obstruction are among the most common sequelae, often leading to persistent respiratory symptoms, impaired lung function, and reduced quality of life long after completion of anti-tuberculosis therapy (3–5).

The coexistence of COPD and previous pulmonary TB has attracted growing attention because both diseases share common risk factors and may interact through several pathophysiological mechanisms. Structural lung damage induced by TB promotes persistent airway inflammation, impaired mucociliary clearance, recurrent respiratory infections, and progressive airflow limitation, thereby increasing susceptibility to COPD development and exacerbations independently of smoking exposure. Previous studies have consistently reported poorer pulmonary function, greater symptom burden, and more frequent exacerbations among COPD patients with a history of pulmonary TB than among those without previous TB (6–9). However, the influence of previous TB on long-term clinical outcomes, particularly exacerbation burden and mortality, remains incompletely understood, especially in TB-endemic countries.

Morocco continues to face a substantial burden of both COPD and tuberculosis; however, data exploring their interaction remain scarce. Therefore, this observational cohort study aimed to evaluate the association between previous pulmonary tuberculosis and clinical outcomes in patients with COPD, with particular emphasis on exacerbation frequency and mortality.

Methods:-

Study design and participants:-

This observational cohort study was conducted in the Department of Pulmonology at Hassan II Hospital, Agadir, Morocco, between January 2022 and March 2024. Consecutive patients aged 40 years or older with a confirmed diagnosis of COPD who were either hospitalized for an acute exacerbation or attended outpatient follow-up visits were eligible for inclusion.COPD was diagnosed according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria, defined by a post-bronchodilator forced expiratory volume in one second (FEV_1)/forced vital capacity (FVC) ratio <0.70 in the presence of compatible respiratory symptoms and relevant risk factor exposure, as confirmed by a pulmonologist (1).Each patient was included only once during the study period. Patients with incomplete medical records, alternative causes of chronic airflow limitation, or unavailable follow-up data were excluded.The study was approved by the Ethics Committee of Mohammed VI University Hospital, Marrakech (Approval No. 124/2024; October 10, 2024). Written informed consent was obtained from all participants before enrollment.

Data collection:-

Demographic characteristics, including age, sex, marital status, educational level, employment status, and socioeconomic status, were recorded. Smoking history was assessed by smoking status, cumulative tobacco exposure (pack-years), smoking cessation duration, and Fagerström nicotine dependence score. Exposure to biomass smoke was also documented.

Clinical variables included COPD duration, body mass index (BMI), modified Medical Research Council (mMRC) dyspnea score, COPD Assessment Test (CAT) score, comorbidities, maintenance respiratory treatment, chronic respiratory failure, and GOLD classification.

Information regarding acute exacerbations included annual exacerbation frequency, severity, presumed etiology, hospitalization, intensive care unit admission, and length of hospital stay. Exacerbations were classified according to the GOLD recommendations as mild, moderate, or severe.Previous pulmonary tuberculosis was defined as a documented history of microbiologically or clinically confirmed pulmonary TB for which anti-tuberculosis treatment had been completed. Radiological sequelae were identified on chest imaging and included fibrotic scars, upper lobe calcifications, traction bronchiectasis, pleural thickening, volume loss, and other chronic destructive changes compatible with previous pulmonary tuberculosis.The duration of COPD was calculated from the date of the first documented diagnosis in the patient's medical records.

Outcomes:-
The primary outcome was the annual frequency of COPD exacerbations according to previous tuberculosis status. Secondary outcomes included exacerbation severity, hospitalization, in-hospital mortality, and overall survival during follow-up.

Statistical analysis:-
Continuous variables are presented as mean ± standard deviation (SD), while categorical variables are expressed as frequencies and percentages. Comparisons between patients with and without a history of pulmonary tuberculosis were performed using appropriate statistical tests according to the type of variable. The association between baseline patient characteristics and long-term mortality was evaluated using a Cox proportional hazards regression model. Variables entered into the multivariable model included age, history of pulmonary tuberculosis, smoking exposure (pack-years), length of hospital stay, annual exacerbation frequency, exacerbation severity, exacerbation etiology, body mass index (BMI), COPD Assessment Test (CAT) score, and chronic respiratory failure. Overall survival after hospital discharge was estimated using the Kaplan–Meier method. Results are presented with 95% confidence intervals (95% CI), and a two-sided P value <0.05 was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics for Windows, version 26.0.

Results:-

Patient characteristics:-
A total of 157 patients with spirometry-confirmed COPD were included in the study. The mean age was 62.5 ± 10.1 years, and the study population was predominantly male (91.1%). Most patients were married (66.9%), had received formal education (59.2%), were currently employed (55.4%), and belonged to a low socioeconomic level (77.1%). Overall, 65 patients (41.4%) had a history of pulmonary tuberculosis. The mean age at TB diagnosis was 12.4 ± 16.7 years. The most common comorbidities were pneumothorax (14.6%), diabetes (8.3%), pulmonary hypertension (7.6%), heart failure (5.1%), and lung cancer (2.5%). The mean duration of COPD was 6.8 ± 5.8 years, and patients experienced a mean of 3.13 ± 1.86 exacerbations per year.

Comparison according to previous pulmonary tuberculosis:-
Baseline demographic and clinical characteristics were generally comparable between patients with and without previous pulmonary tuberculosis (Table 1). No statistically significant differences were observed regarding age, sex, marital status, educational level, employment status, socioeconomic status, COPD duration, GOLD classification, CAT score, chronic respiratory failure, hospitalization, exacerbation severity, or exacerbation etiology.Current smoking was significantly more frequent among patients without previous TB than among those with previous TB (36.3% vs. 21.7%; p = 0.004). Conversely, biomass (wood smoke) exposure was significantly more common among patients with previous TB (0% vs. 5.7%; p = 0.004). Patients with previous TB also experienced a significantly higher annual exacerbation frequency than those without previous TB (p = 0.017). No significant difference was observed in in-hospital mortality between the two groups (1.2% vs. 1.2%; p = 0.607).

Table 1: General characteristics of the patients

	All patients	TB history (-)	TB history (+)	P-value
Subject	**157**	**92**	**65**	
Age (years)	62,5 ± 10,1			0,393
Sex (%)				0,112

Male	91,1	51,6	39,5	
Female	8,9	7	1,9	
Marital status (%)				0,149
Married	66,9	43,3	23,6	
Single	15,3	6,4	8,9	
Divorced	10,2	5,1	5,1	
Widowed	7,6	3,8	3,8	
Education (%)				0,622
Yes	59,2	33,8	25,5	
No	40,8	24,8	15,9	
Work status (%)				0,583
Employed	55,4	32,5	22,9	
Non employed	7	5,1	1,9	
Retired	37,6	21	16,6	
Socioeconomic level (%)				0,673
Medium	22,9	12,7	10,2	
Low	77,1	45,9	31,2	
Underlying disease (%)				
Pneumothorax	14,6	7	7,6	0,256
Diabetes	8,3	5,7	2,5	0,416
HTAP	7,6	5,7	1,9	0,230
Cardiopathy	5,1	2,5	2,5	0,612
Lung cancer	2,5	1,9	0,6	0,5
COPD duration (years)	6,8 ± 5,8			0,285
Smoking history(%)				**0,004**
Current smoker	58	36,3	21,7	
Ex- smoker	36,3	16,6	19,7	
Amount of smoking (pack-years)	32,39 ± 19,91			0,171
Smoking cessation (years)	4,06± 7,49			0,081
Exposure to wood smoke(%)				**0,004**
Yes	5,7	5,7	0	
No	94,3	52,9	41,4	
COPD exacerbation (per year)	3,13 ± 1,86			**0,017**
Ramadan (%)				0,186
Yes	15,3	10,8	4,5	
No	84,7	47,8	36,9	
COPD exacerbation severity (%)				0,089
Mild	5,7	4,5	1,3	
Medium	70,1	43,3	26,8	
Severe	24,2	10,8	13,4	
COPD exacerbation etiology (%)				0,360
Infection	56,7	32,5	24,2	
Poor compliance with treatment	36,3	20,4	15,9	
Treatment cessation				
Tobacco exposure	5,1	3,8	1,3	
	1,9	1,9	0	
GOLD grading (%)				0,438
A	5,7	4,5	1,3	
B	13,4	8,3	5,1	
E	80,9	45,9	35	
Hospitalization (%)				0,319
Yes	80,9	45,9	35	
No	19,1	12,7	6,4	
Duration (days)	5,48 ± 3,06			0,505
BMI (kg/m2) (%)				0,390

Underweight	35,7	23,6	12,1	
Normal	42	24,2	17,8	
Overweight	16,6	7,6	9	
Obese	5,7	3,2	2,5	
CAT score (%)				0,449
<10	6,4	4,5	1,9	
≥10	93,6	54,1	39,5	
Chronic respiratory failure (%)				0,632
Yes	15,3	8,3	7	
No	84,7	50,3	34,4	
Evolution (%)				0,607
ICU transfer rate	7	3,2	3,8	
In-hospital mortality	2,5	1,2	1,2	

Survival analysis:-

Patients were followed until March 2025, corresponding to one year after the inclusion of the last participant. Kaplan–Meier analysis showed that the one-year survival rate was significantly lower among patients with a history of pulmonary tuberculosis than among those without previous TB (87.7% vs. 94.6%, respectively).The overall median survival was 36 months (± 2.2 months; 95% CI: 31.7–40.3 months). Patients with a history of pulmonary tuberculosis had a shorter median survival of 36 months (± 2.6 months; 95% CI: 31.0–41.0 months), whereas those without previous TB had a median survival of 48 months (± 4.3 months; 95% CI: 39.6–56.4 months).

Comparison of survival distributions using the Kaplan–Meier method demonstrated a statistically significant difference between the two groups (log-rank $\chi^2 = 4.396$; $p = 0.036$). Similar findings were obtained with the Breslow ($p = 0.050$) and Tarone–Ware ($p = 0.044$) tests, indicating that patients with a history of pulmonary tuberculosis had poorer long-term survival than those without previous TB (Figure 1).

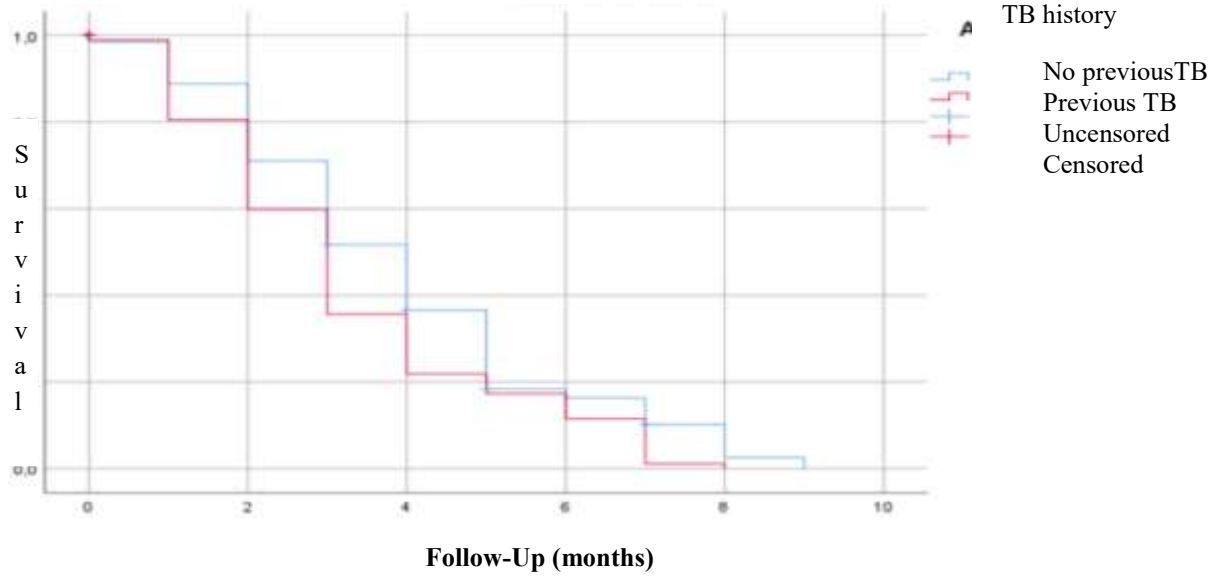

Figure 1 Kaplan–Meier curve showing the 1-year survival of the COPD patients with and without TB history (P=0.036).

Abbreviation: TB, tuberculosis:-

In the multivariable Cox proportional hazards regression analysis, the annual number of COPD exacerbations was independently associated with long-term mortality. In contrast, a history of pulmonary tuberculosis was not independently associated with mortality after adjustment for potential confounding factors (Table 2).

Table 2: Factors that affect mortality according to Cox analysis

	HR	95% CI	p value
Age	0,988	0,965-1,012	0,340
Tuberculosis History	1,634	0,559-4,774	0,369
Smoking History (Pack-years)	0,998	0,989-1,008	0,736
Annual number of COPD exacerbations	0,299	0,207-0,432	**<0,001**
COPD exacerbation severity	1,017	0,608-1,699	0,950
COPD exacerbation etiology	1,147	0,838-1,569	0,392
Length of hospital stay	1,024	0,898-1,168	0,725
BMI	0,892	0,712-1,118	0,322
CAT score	0,672	0,152-2,959	0,599
Chronic respiratory failure	0,864	0,319-2,345	0,775

Discussion:-

The present study investigated the impact of previous pulmonary tuberculosis on clinical outcomes in patients with COPD. Our findings demonstrate that patients with a history of pulmonary tuberculosis experienced a significantly higher annual frequency of COPD exacerbations compared with those without previous TB. However, after adjustment for potential confounding factors, previous tuberculosis was not independently associated with long-term mortality. These findings suggest that previous pulmonary tuberculosis contributes primarily to increased disease burden and exacerbation susceptibility rather than directly influencing survival.

The relationship between pulmonary tuberculosis and chronic obstructive pulmonary disease has gained increasing attention over the past decade. Although cigarette smoking remains the principal risk factor for COPD, previous pulmonary tuberculosis has emerged as an additional determinant of chronic airflow limitation and poor respiratory outcomes, particularly in countries where TB remains endemic. Tuberculosis induces irreversible structural damage to the lung parenchyma and airways that frequently persists despite microbiological cure, giving rise to the clinical entity now recognized as post-tuberculosis lung disease (PTLD). PTLD encompasses persistent respiratory symptoms, chronic airflow obstruction, impaired exercise capacity, and radiological sequelae that substantially affect long-term respiratory health (3,10).

Our results are consistent with those reported by Park et al., who demonstrated that COPD patients with previous pulmonary tuberculosis exhibited more severe airflow limitation, poorer health-related quality of life, and a significantly higher frequency of acute exacerbations than patients without TB history (6). Their study suggested that residual structural lung damage following tuberculosis accelerates COPD progression by increasing susceptibility to recurrent respiratory infections and chronic airway inflammation. Similarly, the significantly higher exacerbation frequency observed in our cohort supports the concept that previous pulmonary tuberculosis defines a distinct COPD phenotype characterized by increased clinical instability.

Several pathophysiological mechanisms may explain this association. Pulmonary tuberculosis frequently results in permanent anatomical abnormalities, including parenchymal fibrosis, volume loss, bronchial distortion, pleural thickening, cavitary lesions, and traction bronchiectasis. These structural alterations impair mucociliary clearance, promote mucus retention, and facilitate persistent bacterial colonization of the lower airways. Chronic bacterial colonization, in turn, maintains low-grade airway inflammation, perpetuates neutrophilic inflammatory responses, and increases susceptibility to recurrent respiratory infections, which represent the principal trigger of COPD exacerbations. Moreover, chronic destruction of the pulmonary microvasculature and small airways contributes to progressive airflow limitation that may continue long after completion of anti-tuberculosis treatment (3,9,10).
Radiological evidence strongly supports these mechanisms. Jin et al. demonstrated that COPD patients with previous pulmonary tuberculosis exhibited a significantly higher prevalence of emphysema and bronchiectasis on computed tomography than COPD patients without TB history (8).

Importantly, bronchiectasis was associated with more severe airflow obstruction, poorer pulmonary function, and increased exacerbation frequency. These findings provide structural evidence supporting the higher exacerbation burden observed in our study and emphasize the importance of systematic radiological evaluation in COPD patients with previous tuberculosis.

Persistent impairment of lung function following successful tuberculosis treatment has also been consistently documented. Using data from the Korean National Health and Nutrition Examination Survey, Jung et al. reported significantly lower FEV_1 values and FEV_1/FVC ratios among tuberculosis survivors, even after adjustment for smoking and other confounding factors (7). These findings indicate that pulmonary tuberculosis may produce irreversible airflow obstruction independent of tobacco exposure, thereby predisposing affected individuals to COPD development and progression. Similar conclusions have been reached by several recent studies demonstrating that pulmonary function impairment often persists for years after microbiological cure and represents one of the principal components of PTLD (3,5,10).

Interestingly, despite the significantly higher exacerbation frequency among patients with previous tuberculosis, TB history was not independently associated with mortality after multivariable adjustment. Instead, the annual number of COPD exacerbations emerged as the principal determinant of long-term mortality. This finding is biologically plausible because recurrent exacerbations accelerate the decline in lung function, amplify systemic inflammation, increase cardiovascular complications, and contribute to progressive respiratory failure. Therefore, tuberculosis may influence prognosis indirectly by increasing exacerbation burden rather than acting as an independent predictor of mortality. Similar observations have been reported in previous COPD cohorts, in which exacerbation frequency consistently predicted mortality more strongly than baseline etiological factors.

Our Kaplan–Meier analysis demonstrated a significant difference in survival between patients with and without previous pulmonary tuberculosis. However, this association disappeared after adjustment in the multivariable Cox proportional hazards model. The apparent discrepancy between these analyses suggests that the survival difference observed in the unadjusted analysis may largely reflect differences in disease severity and exacerbation burden rather than a direct effect of tuberculosis itself. In addition, the relatively limited sample size and the low number of deaths during follow-up may have reduced the statistical power required to detect an independent association between previous tuberculosis and mortality.

Another interesting observation was the significantly lower prevalence of current smokers among patients with previous pulmonary tuberculosis despite their higher exacerbation frequency. This finding suggests that structural pulmonary damage induced by tuberculosis may represent an independent contributor to COPD progression, irrespective of cumulative tobacco exposure. Clinicians should therefore avoid underestimating disease severity in COPD patients with previous tuberculosis simply because of a lower smoking burden. In regions with a high prevalence of tuberculosis, previous TB should be regarded as an additional respiratory risk factor that may influence long-term disease evolution independently of smoking.

The present study has several strengths. To our knowledge, it represents one of the few studies from North Africa investigating the influence of previous pulmonary tuberculosis on COPD outcomes. Furthermore, both hospitalized patients and those managed in the outpatient setting were included, providing a broad representation of disease severity encountered in routine clinical practice. Clinical evaluation was performed using standardized COPD assessment tools, and long-term survival was assessed using both Kaplan–Meier analysis and multivariable Cox regression.

Several limitations should nevertheless be acknowledged. First, the single-center design and relatively modest sample size may limit the generalizability of our findings. Second, although patients were prospectively enrolled, residual confounding cannot be completely excluded despite multivariable adjustment. Third, quantitative chest CT analysis was unavailable, preventing detailed assessment of emphysema severity, bronchiectasis extent, and fibrotic destruction. Likewise, microbiological evaluation of chronic airway colonization and inflammatory biomarkers was not performed, precluding further investigation of the mechanisms underlying recurrent exacerbations. Finally, the duration of follow-up may have been insufficient to fully evaluate the long-term impact of previous tuberculosis on mortality.

Overall, our findings provide additional evidence that previous pulmonary tuberculosis identifies a clinically relevant COPD phenotype characterized by increased exacerbation frequency. Recognition of post-tuberculosis lung disease is particularly important in countries where tuberculosis remains highly prevalent. Early identification of COPD patients with previous TB may allow closer clinical follow-up, optimization of inhaled treatment, pulmonary rehabilitation, smoking cessation support, vaccination, and prompt management of respiratory infections to reduce future exacerbations. Future multicenter prospective studies with larger cohorts, longer follow-up, and

comprehensive radiological and functional assessment are needed to better define the independent contribution of previous tuberculosis to COPD progression and long-term survival.

Conclusion:-

Previous pulmonary tuberculosis is associated with an increased frequency of COPD exacerbations, underscoring the lasting impact of post-tuberculosis lung damage on disease progression. Although previous TB was not independently associated with mortality after adjustment for confounding factors, exacerbation frequency emerged as the strongest predictor of long-term survival. These findings support the recognition of post-tuberculosis lung disease as a distinct COPD phenotype requiring closer clinical surveillance and individualized management, particularly in tuberculosis-endemic regions. Incorporating a history of pulmonary tuberculosis into routine COPD assessment may help identify patients at increased risk of exacerbations and guide targeted preventive strategies, including optimization of inhaled therapy, pulmonary rehabilitation, vaccination, and prompt management of respiratory infections. Further large-scale prospective multicenter studies are needed to clarify the biological mechanisms underlying post-tuberculosis lung disease and to determine its long-term impact on COPD progression and prognosis.

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