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The impact of smoking on the severity of COPD

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Abstract

Chronic obstructive pulmonary disease (COPD) is a major global health burden and remains strongly associated with tobacco exposure. This retrospective study evaluated the impact of active and past smoking on the clinical and functional severity of COPD within a Moroccan cohort. Data were collected from 70 patients managed in the pulmonology department of Mohammed VI University Hospital in Marrakesh from July 2024 to July 2025. The mean age was 64 years, with a marked male predominance (88%). Active smokers accounted for 67% of the cohort, with an average cumulative exposure of 36 pack-years. According to GOLD classification, severe and very severe stages (III–IV) were significantly more frequent among active smokers (74%) compared with former smokers (42%) and non-smokers (29%). Active smokers also exhibited higher dyspnea scores and a greater number of severe exacerbations requiring hospitalization. A significant correlation was observed between cumulative tobacco exposure and the degree of airflow limitation ($p < 0.01$), consistent with established evidence linking smoking intensity to accelerated FEV1 decline. The persistence of severe disease in some former smokers highlights the role of additional determinants, including genetic predisposition, occupational hazards, and environmental pollution—particularly biomass exposure, which may explain the 10% of non-smokers with COPD in this cohort. These findings underscore the need for systematic assessment of smoking status and exposure profiles in COPD management and emphasize the importance of smoking cessation, while encouraging further research integrating genetics, biomarkers, advanced imaging, and AI-based phenotyping to better understand COPD heterogeneity within the Moroccan population.

Keywords: COPD; Smoking; Airflow limitation; FEV1 decline

1. Introduction

Chronic obstructive pulmonary disease (COPD) is a chronic respiratory condition characterized by progressive and partially reversible airflow limitation. It is currently the third leading cause of death worldwide and a major public health issue. Smoking is the main risk factor, accounting for 80 to 90% of cases in industrialized countries.

The pathophysiology is based on chronic inflammation of the airways and lung parenchyma, exacerbated by exposure to tobacco smoke. This inflammation leads to bronchial remodeling, goblet cell hyperplasia, increased mucus production, impaired mucociliary transport, and progressive destruction of the alveolar walls (emphysema). These mechanisms result in fixed bronchial obstruction, aggravated by repeated acute exacerbations.

Our objective is to analyze, within a Moroccan cohort, the impact of active and past smoking on the clinical and functional severity of COPD.

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2. Patients and methods

This is a retrospective study of patients treated for COPD in the pulmonology department of Mohammed VI University Hospital in Marrakesh over a 12-month period (July 2024–July 2025).

3. Results and discussion

We collected data on 70 patients with COPD over a 12-month period. The average age was 64 (range 42–82), with a clear male predominance (88%), reflecting the classic distribution of the disease, which is strongly correlated with male smoking in our sociocultural context.

Regarding smoking status, 67% were active smokers, 23% were former smokers, and 10% were non-smokers. The average smoking load was 36 pack-years. This high value reflects significant cumulative exposure, known to be the main determinant of the onset and progression of COPD.

Functional severity according to the GOLD classification showed that 8% of patients were in stage 1, 31% in stage 2, 38% in stage 3, and 23% in stage 4. There was a clear predominance of severe and very severe forms (stages 3 and 4) among active smokers (74%) compared to former smokers (42%) and non-smokers (29%). This distribution perfectly illustrates the cumulative and continuous effect of smoking on the decline in FEV1.

In terms of symptoms, dyspnea was the main symptom (92%), followed by chronic productive cough (61%) and frequent exacerbations (47%). Active smokers not only had higher dyspnea scores (mMRC ≥ 2), but also an increased frequency of severe exacerbations requiring hospitalization, confirming the deleterious effect of smoking on clinical progression.

The correlation between cumulative smoking exposure and the severity of bronchial obstruction ($p < 0.01$) reinforces the data in the international literature. Several longitudinal studies, including the Lung Health Study cohort, have shown that the annual decline in FEV1 is directly proportional to tobacco consumption. The mechanism is based on persistent neutrophilic inflammation, increased oxidative stress, activation of proteases (elastases, metalloproteinases), and inhibition of anti-proteases, leading to progressive destruction of the lung parenchyma.

An important point is that even after quitting smoking, some patients progress to severe forms of the disease. This highlights the fact that, while smoking cessation slows the decline in FEV1, it does not reverse the irreversible structural damage that has already occurred. This observation highlights the role of other factors in the progression of COPD: genetic predisposition (e.g., alpha-1-antitrypsin deficiency), occupational exposure (dust, silica, welding fumes), air pollution, and biomass combustion (particularly common in rural areas of Morocco).

Thus, in our cohort, the significant proportion of non-smokers with COPD (10%) is probably due to environmental or occupational exposure, a well-documented phenomenon in developing countries.

In clinical practice, these results reinforce the need to systematically include an assessment of smoking status and exposures in the management of COPD, as well as to promote smoking cessation through appropriate programs, including nicotine replacement therapy, behavioral therapies, and possibly pharmacotherapy (varenicline, bupropion).

4. Conclusion

Active smoking is a major determinant of COPD severity, with a clear dose-dependent relationship between smoking exposure and worsening bronchial obstruction. Smoking cessation remains a fundamental measure for slowing functional decline and reducing the frequency of exacerbations, but it alone does not explain the interindividual variability observed in the progression of the disease. This clinical and functional heterogeneity suggests the involvement of additional factors, such as genetic predisposition and occupational or environmental exposures (biomass combustion, urban pollution). A key question therefore remains: how can we more accurately characterize the interaction between smoking, individual susceptibility, and these exposures in the genesis and progression of COPD, particularly within the Moroccan population? Future research based on multidimensional approaches, combining genetics, inflammatory biomarkers, high-resolution imaging, and artificial intelligence applied to phenotyping, could identify specific risk profiles and pave the way for truly personalized prevention and management strategies.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

The present research work does not contain any studies performed on animals/humans subjects by any of the authors'.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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