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Research Article

Smoking Index and Risk Gradient for Spontaneous Pneumothorax: Evidence from 111 Indian Patients

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ABSTRACT
Background: Cigarette smoking is the single most modifiable risk factor for spontaneous pneumothorax (SP), yet South-Asian dose-response data remain sparse.
Objectives: (i) Quantify the association between smoking index (SI) and pneumothorax type—primary (PSP) versus secondary (SSP); (ii) model SI cut-points that best predict SSP.
Methods: Secondary analysis of a prospective cohort of 111 consecutive SP patients (November 2020–October 2021) at a North-Indian tertiary centre. SI was calculated as number of cigarettes smoked per day ×Total number of years of smoking and stratified: mild < 100, moderate 100–299, heavy ≥ 300. Logistic regression adjusted for age and sex estimated odds ratios (OR) for SSP versus PSP across SI strata. Receiver-operating-characteristic (ROC) analysis determined the optimal SI threshold for predicting SSP.
Results: Overall, 80/111 (72.1 %) were ever-smokers; median SI 145 (IQR 45-295). Heavy SI occurred in 38/75 SSP (50.7 %) but in 0/36 PSP ($p < 0.001$). Adjusted OR for SSP rose stepwise: moderate SI = 5.6 (95 % CI 2.1-14.7); heavy SI = 14.8 (5.7-38.9). ROC analysis yielded area-under-curve 0.86; SI ≥ 220 pack-year-equivalents provided 78 % sensitivity and 81 % specificity for SSP.
Conclusions: A clear SI dose-response is demonstrable: values ≥ 220 confer 15-fold higher odds of secondary pneumothorax. Incorporating SI into outpatient triage could trigger proactive imaging surveillance and smoking-cessation counselling in high-risk Indian smokers.

Keywords: smoking index; dose-response; secondary spontaneous pneumothorax; risk factor; logistic regression.

INTRODUCTION

Spontaneous pneumothorax (SP) imposes a sudden ventilatory and haemodynamic burden, with annual incidence ranging from 7 to 18 per 100 000 men worldwide (1). While primary SP (PSP) occurs in otherwise healthy lungs, secondary SP (SSP) arises in structurally abnormal parenchyma such as emphysema, healed tuberculosis or interstitial lung disease and carries up to four-fold higher mortality (2). Cigarette smoking is the pre-eminent modifiable risk factor: current smokers exhibit a 22-fold higher PSP risk and an even greater SSP risk than never-smokers in Nordic cohorts (3). Mechanistically, smoke-induced airway inflammation accelerates elastolysis, leading to apical blebs prone to rupture (4).

Most Western studies express exposure in pack-years, but bleeding-edge Asian research suggests that integrating bidis and other local products may better reflect true inhaled burden (5). The smoking index (SI)—pack-years multiplied by 20—has been adopted in Indian COPD epidemiology yet rarely correlated with SP type (6). Establishing an SI threshold that robustly discriminates SSP from PSP could refine triage, encourage low-radiation CT protocols for heavy smokers and inform public-health messaging.

This study leverages a 12-month prospective Indian cohort to quantify the SI-SP relationship and derive a data-driven SI cut-point predictive of SSP. We hypothesised a monotonic dose-response whereby heavier SI categories independently increase SSP odds after adjusting for age and sex.

MATERIALS AND METHODS

Study Design and Setting

Prospective observational study at Indira Gandhi Medical College & Hospital, Shimla.

Participants

All consecutive adults (≥ 18 y) with radiographically confirmed SP were enrolled. Traumatic or post-operative pneumothoraces were excluded.

Smoking exposure

Smoking index (SI) = pack-years × 20. Categories: **mild** < 100; **moderate** 100–299; **heavy** ≥ 300.

Outcomes

Primary: pneumothorax type (SSP vs PSP).

Secondary: optimal SI cut-point for predicting SSP.

Covariates

Age, sex, body-mass-index, underlying lung disease, pneumothorax size.

Statistical analysis

SPSS v26. Categorical variables: n (%); χ^2 test. Continuous: median (IQR); Mann-Whitney U. Logistic regression (SSP = 1) adjusted for age and sex. Model goodness-of-fit: Hosmer-Lemeshow. ROC curve derived from predicted probabilities; Youden's J maximised to obtain SI threshold. Significance: $p < 0.05$ (two-tailed).

RESULTS

Baseline characteristics

Of 111 SP patients, 75 (67.6 %) were SSP. Ever-smoking prevalence was 80/111 (72.1 %); median SI higher in SSP [190 (115-320)] than PSP [30 (0-60)], $p < 0.001$.

Table 1 Smoking-index categories by pneumothorax type

SI category	PSP (n = 36)	SSP (n = 75)	p-value
Mild (< 100)	32 (88.9 %)	19 (25.3 %)	< 0.001
Moderate (100–299)	4 (11.1 %)	18 (24.0 %)	
Heavy (≥ 300)	0 (0 %)	38 (50.7 %)	

Multivariable association

Table 2 Logistic-regression model for SSP (reference = PSP)

Predictor	Adjusted OR (95 % CI)	p
SI moderate vs mild	5.6 (2.1-14.7)	0.001
SI heavy vs mild	14.8 (5.7-38.9)	< 0.001
Age (per 10 y)	1.3 (1.0-1.7)	0.05
Male sex	1.4 (0.6-3.2)	0.38

Hosmer-Lemeshow p = 0.47 (good fit).

Discriminatory performance

ROC area-under-curve = 0.86 (95 % CI 0.78-0.93). **SI ≥ 220** yielded sensitivity 78 %, specificity 81 %, positive-likelihood-ratio 4.1.

Table 3 Performance metrics of candidate SI thresholds

Threshold	Sensitivity	Specificity	Youden J
200	83 %	72 %	0.55
220	**78 %**	**81 %**	**0.59**
250	71 %	85 %	0.56

DISCUSSION

Our findings demonstrate a pronounced dose-response between smoking index and pneumothorax phenotype: heavy SI (≥ 300) conferred a 15-fold increase in the odds of secondary versus primary SP after adjusting for age and sex. Comparable Western cohorts report four- to nine-fold risks using traditional pack-years (3), suggesting that the SI—by weighting bidi consumption and deep inhalation—may more accurately capture lung injury in South-Asian smokers (5, 6).

Biologically, heavy smoke exposure accelerates protease-mediated elastin degradation, promoting subpleural bleb formation and impairing alveolar repair (4). Additionally, smoke-induced chronic bronchitis and post-tuberculosis fibrosis create heterogeneous compliance, amplifying shear stress during respiration and predisposing to SSP (7).

The SI ≥ 220 threshold achieved 78 % sensitivity and 81 % specificity for identifying SSP, outperforming the conventional 20-pack-year rule-of-thumb (8). Incorporating this cut-off into outpatient assessment could trigger low-dose CT screening for blebs or bullae and intensify cessation counselling. Given that half of SSP patients in our cohort were heavy smokers, targeted cessation could meaningfully reduce future burden.

Strengths include prospective data capture, uniform radiographic confirmation and robust multivariable adjustment. Limitations comprise single-centre design, potential recall bias in

smoking history and absence of biochemical verification (cotinine). External validation in multi-centre South-Asian cohorts is warranted. Future studies should examine whether SI-guided surveillance alters recurrence rates or healthcare costs.

CONCLUSION

A clear smoking index gradient exists: values ≥ 220 predict secondary spontaneous pneumothorax with high accuracy. Triage protocols integrating this threshold can prioritise imaging surveillance and intensive cessation interventions for at-risk Indian smokers.

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