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Recovery after Ordeal: Respiratory Alterations Linked to Quitting Smoking

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Abstract

Background: Smoking cessation has been long considered a sole precautionary mode of respiratory disability in both developed and developing countries. **Aim of Work:** our study aims to assess the impact of smoking cessation on clinical features as well as pulmonary functional measurement and exhaled CO level in ex-smokers. **Patients and Methods:** This observational cross sectional study was conducted on 306 subjects who were attending to outpatient pulmonology clinic in Center of medical services in Taibah University at Al-Medina Al-Munwara, Kingdom of Saudi. Subjects included in this study were selected as being ex-smoker for more than 6 months. They were classified according to the duration of smoking cessation into a) recent smoking quitter (stopped smoking ≤2 years) and; b) remote smoking quitter (stopped smoking >2 years). They were subjected to clinical evaluation, spirometry and exhaled CO level measurement in exhaled air. **Results**: All clinical features including cough, dyspnea, chest pain, hemoptysis, wheezes, hospitalization and relapse in both recent quitter and remote quitter subjects were statistically comparable but without statistical significant difference. There was statistical positive correlation between duration of smoking cessation and actual FEV1, actual FVC, actual PEFR, actual TLC, functional defect FEV1, functional defect of FVC, functional defect of PEFR and functional defect of TLC while statistical negative correlation was with CO exhaled level. **Conclusion:** Smoking cessation demonstrated an appreciated role in recovering patient clinical as well as pulmonary functional and gaseous characters towards healthy side.

Keywords

Smoking Cessation, FEV1, FVC, Exhaled CO, Relapse, Hospitalization

1. Introduction

Thus far, smoking is the sole prime needless cause of death and disability in developed countries. Smoking rate is now rising rapidly all over the world and is one of the prevailing threats to world health both now and ongoing [1]. Most smokers, could not gain the will to quit smoking in spite of being is the only most vital thing they hardly require to improve their health. Boosting smoking cessation is one of the most real and cost effective possessions that doctors and other health professionals can acquire to improve health and prolong their patients' lives. Frequently, the uptake and consequential adverse effects of smoking occur earlier and to a greater grade among male sex [2].

Smoking has various recognized adverse health effects, and has been comprehensively investigated and documented. Dose-response relationship with degree of smoking has been reported, duration of smoking, and early interest associated with higher risks of smoking related respiratory troubles and mortality. The commonest cause-specific links are with respiratory cancers and chronic obstructive pulmonary disease; in numeric terms, the utmost health influences of smoking are on respiratory and cardiovascular diseases. Cessation of smoking has considerable immediate and delayed health profits for smokers for all age groups. The much more hazard of demise from smoking occurs immediately after cessation and continues to do so for 10 - 15 years at least. Former smokers live longer than persistent smokers, no matter what age they stop smoking, though the influence of quitting on mortality is greatest at younger ages [1] [3]. The rate and extent of minimization of risk vary between diseases—for lung cancer it ranges from 10 years to about 30% - 50% that of persistent smokers, but the risk remains high still after 20 years of quitting. There is benefit from cessation at all ages, but before age 30 eradicates 90% of the ultimate risk of lung cancer [4]. Smoking is associated with hastened rate of deterioration in lung function with age. Cessation results in a minor improvement in lung function and reverses the effect on ensuing rate of decline, which reverts to that in non-smokers. Thus, prompt cessation is especially important in liable persons to preclude or postpone the onset of chronic obstructive pulmonary disease, mortality and symptoms are reduced in former smokers compared with lasting smokers [5].

Our study aim in this study is to assess the impact of smoking cessation on clinical features as well as pulmonary functional measurement and exhaled CO in ex-smokers attending outpatient pulmonology clinic in Center of medical services in Taibah University in Al-Medina Al-Munwarah.

2. Patients and Methods

This study was conducted on 306 subjects who attended to outpatient pulmo-

nology clinic in Center of medical services in Taibah University at Al-Medina Al-Munwarah, Kingdom of Saudi Arabia (from August 2018 till May 2019), with age ranging from 22 to 68 years old. Subjects included in this study were selected as being ex-smoker more than 6 months.

2.1. Study Design

This is an observational cross sectional study in which smoking quitters were adopted and included by a screening method for patients with clinical as well as functional parameters with exhaled CO level changes after smoking cessation.

2.2. Patients

Inclusion criteria:

Ex-smokers as those who had not smoked for at least six months [6]. Whatever the method adopted in cessation program were defined and enrolled with informed consent.

Classification: They were subdivided into

According smoking index [7]

Mild quitter: 258 *subjects* (84.3%)

Moderate quitter: 36 *subjects* (11.8%)

Heavy quitter: 12 (3.9%)

Pack-Year index = *No of cigarette/ day* × *duration in years*/20 [8]

Mild: *less than* 20 *pack-year*

Moderate: 20 - 49 *pack-year*

Heavy: more than 49 *pack-year*

Hookah (***Korsi/ day***) [7]

Mild: *less than* 10 *Korsi/ day*

Moderate: 10 - 20 *Korsi/ day*

Severe: more than 20 *Korsi/ day*

According duration of smoking cessation [9]

Recent smoking quitter (*stopped smoking* ≤ 2 *years*): 126 (41.2%)

Remote smoking quitter (*stopped smoking* > 2 *years*): 180 (58.8%)

According type of smoking

Cigarette smokers 204 (66.7%)

Hookah smokers 66 (21.6%)

Mixed smokers 24 (7.8%)

Others (*vape*) 12 (3.9%)

Exclusion criteria:

1) Organ failure
2) Bronchial asthma
3) Severe COPD cases
4) Occupational lung diseases
5) Pulmonary eosinophilia
6) Pneumonia
7) Interstitial lung disease

8) Heart failure

2.3. Methods

1) Clinical evaluation including history taking and clinical examination: including, age, occupation, present history including cough, dyspnea, chest pain, haemoptysis, past history especially history of pulmonology department admission, family history pulmonary diseases.

2) Investigations:

➢ Radiographic: Chest X ray (PA & lateral views).

3) Spirometry

Pulmonary function test: Baseline pulmonary function testing, using "Smart PFT CO" device (Figure 1). Manufactured by medical equipment Europe—Hammelburg—Germany

4) Exhaled CO measurement: CO detector check plus

All subjects were submitted to measurement of exhaled carbon monoxide by **Co detector check plus,** Tracy Rowland—Dorset Public Health, England. By **MD Diagnostics Ltd.** 15 Hollingworth Court, Turkey Mill, Ashford Road, Maidstone, Kent ME14 5PP in Figure 2. Subjects were advised to breathe into a

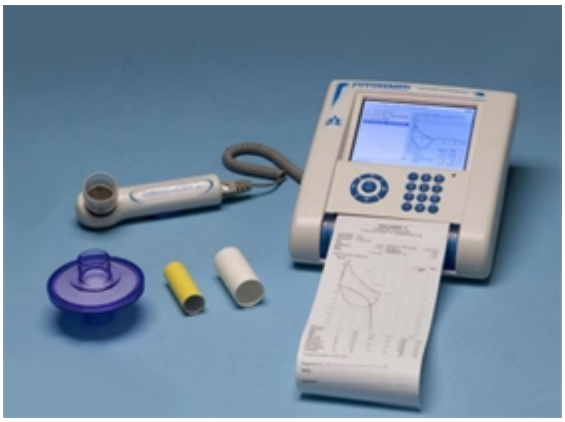

Figure 1. Discovery 2, Future Med. portable Spirogram (Futuremed America. Inc., 15700 Devonshire St., Granada Hills, CA 91344-7225, USA).

Figure 2. CO check device.

cardboard tube attached to a handheld monitor. The monitor then showed the reading on its screen. They were asked to hold your breath for 10 - 15 seconds. Then they breathe out slowly into the mouthpiece aiming to empty their lungs completely. The results were recorded in parts per million (ppm) of carbon monoxide in breath.

Values were detected according British lung foundation 2017 for exhaled level: [10]

- If the level of exhaled CO level ≥ 10 ppm → Recent exposure to a high level of carbon monoxide = you are a smoker.
- If the level of exhaled CO level 5 - 9 ppm → Recent exposure to a moderate level of carbon monoxide = you are possibly smoking.
- If the level of exhaled CO level 1 - 4 ppm → Recent exposure to a low level of carbon monoxide. It's normal to have a small amount of carbon monoxide in your breath even if you're not a smoker.

3. Results

Our study was conducted on 306 subjects (Table 1) who were classified according to smoking index into mild quitters [258 (84.3%)], moderate quitters [36 (11.8%)] and heavy quitters [12 (3.9%)]. There was a statistical significant difference between them which was in favor to mild quitter. According to duration of smoking cessation, subjects were classified into recent quitters [126 (41.2%)] and remote quitters [180 (58.8%)]. There was no statistical significant difference between them. According to type smoking, subjects were classified into cigarette smokers [204 (66.7%)], hookah smokers [66 (21.6%)], mixed smokers [24 (7.8%)] and other smokers [12 (3.9%)]. There was a statistical significant difference between them which was in favor to cigarette smokers.

Demographic features of studied subjects classified according to duration of smoking cessation

There was no statistical significant difference in the mean values of age (p =

Table 1. Classification of studied subjects.

Classification	Type	Number (%)	p value
According smoking index	Mild quitter	258 (84.3%)	0.001*
	Moderate quitter	36 (11.8%)	
	Heavy quitter	12 (3.9%)	
According duration of smoking cessation	Recent quitter	126 (41.2%)	0.208
	Remote quitter	180 (58.8%)	
According type of smoking	Cigarette smokers	204 (66.7%)	0.001*
	Hookah smokers	66 (21.6%)	
	Mixed smokers	24 (7.8%)	
	Others (Vape)	12 (3.9%)	

p > 0.05 = not significant. *p ≤ 0.05 = significant.

0.760), duration of smoking (p = 0.928) and duration of cessation (p = 0.068) between recent quitters and their corresponding levels in remote quitter subjects. As regards occupation the distribution of employee and students in both recent quitter subjects and remote quitter subjects were statistically comparable (p = 0.304).

All clinical findings including cough, dyspnea, chest pain, hemoptysis, wheezes, hospitalization and relapse in both recent quitter subjects and remote quitter subjects were statistically comparable. Clinical features showed higher percentage in remote quitter than recent quitter.

There was no statistical significant difference in clinical findings between both recent and remote quitter subjects including cough (p = 0.688), dyspnea (p = 0.718), chest pain (p = 0.796), wheezes (p = 0.293), hospitalization (p = 0.634) and relapse (p = 0.159).

Comparison between mean values of pulmonary function parameters in study subjects classified according to duration of smoking cessation

There was no statistical significant difference in the mean values of different pulmonary function parameters between recent quitter subjects and their corresponding levels in remote quitter subjects.

All pulmonary functional parameters illustrated in Table 2 showed higher levels and better measurement in remote quitter than recent quitter but without statistical significance differences.

Table 2. Pulmonary functional data among classified groups.

	Recent quitter (n = 126)	Remote quitter (n = 180)	p value
Predicted FEV1	3.32 ± 0.06	3.33 ± 0.21	0.685
Actual FEV1	2.87 ± 0.60	3.12 ± 0.76	0.225
Functional defect of FEV1	0.97 ± 0.94	0.89 ± 0.87	0.804
Predicted FVC	4.18 ± 0.27	4.16 ± 0.28	0.837
Actual FVC	3.42 ± 0.60	3.71 ± 0.66	0.120
Functional defect of FVC	0.79 ± 0.45	0.69 ± 0.21	0.416
Predicted FEV1/FVC	79.29 ± 4.76	79.93 ± 3.87	0.595
Actual FEV1/FVC	82.71 ± 9.54	83.17 ± 10.22	0.874
Functional defect of FEV1/FVC	0.09 ± 0.04	0.13 ± 0.07	0.238
Predicted PEFR	9.67 ± 0.45	9.86 ± 0.64	0.245
Actual PEFR	6.86 ± 2.80	7.35 ± 2.58	0.519
FFD of PEFR	3.32 ± 2.42	2.95 ± 2.00	0.576
Predicted TLC	5.29 ± 0.29	5.16 ± 0.58	0.337
Actual TLC	4.33 ± 0.68	4.70 ± 0.77	0.090
FFD of TLC	1.08 ± 0.69	0.86 ± 0.28	0.201
CO exhaled level	1.52 ± 0.60	1.57 ± 0.97	0.858
SpO_2	96.52 ± 1.17	96.70 ± 1.06	0.577

Data are expressed as mean ± SD or number (%). p > 0.05 = not significant.

There were statistical positive correlations between duration of smoking cessation and actual FEV1 (r = 0.535; p = 0.702), actual FVC (r = 0.388; p = 0.299), actual PEFR (r = 0.415; p = 0.292), actual TLC (r = 0.548; p = 0.302), functional defect FEV1 (r = 0.624; p = 0.001), functional defect of FVC (r = −0.117; p = 0.461), functional defect of PEFR (r = 0.100; p = 0.517) and functional defect of TLC (r = −0.183; p = 0.257) while statistical negative correlation between CO exhaled level (r = −0.119; p = 0.406).

Table 3 showed that mild quitter had lowest clinical features among other groups aside from haemoptysis, chest pain and wheezes that were absent in all groups except chest pain that presented very low percentage < 1%.

Table 4 showed that all clinical features were higher in mixed group than other groups but without significant statistical differences.

All functional parameters were higher in mild than moderate than heavy quitter with significant statistical differences regarding FEV1 (predicted-actual and functional defect), actual PEFR, actual FVC.

Both actual FEV1, and FVC were more in group other followed by hookah

Table 3. Clinical data among of patients classified according to smoking index subgroups.

	Mild quitter (n = 258)	Moderate quitter (n = 36)	Heavy quitter (n = 12)	p value
Cough (yes)	18 (41.9%)	6 (100.0%)	2 (100.0%)	0.002
Dyspnea (yes)	8 (18.6%)	6 (100.0%)	2 (100.0%)	0.062
Chest pain (yes)	2 (0.77%)	0 (0.0%)	0 (0.0%)	-
Haemoptysis (yes)	0 (0.0%)	0 (0.0%)	0 (0.0%)	-
Wheezes (yes)	0 (0%)	0 (0.0%)	0 (0.0%)	-
Repeated Hospitalization (yes)	2 (4.7%)	0 (0.0%)	2 (100.0%)	-
Relapse	1.49 ± 1.37	1.00 ± 0.63	3.50 ± 0.71	0.056

Data are expressed as mean ± SD or number (%).

Table 4. Clinical data among of patients classified according to type of smoking subgroups.

	Cigarette (n = 204)	Hookah (n = 66)	Mixed (n = 24)	Others (n = 12)	p value
Cough (yes)	18 (52.9%)	4 (36.4%)	4 (100%)	0 (0.0%)	0.325
Dyspnea (yes)	8 (23.5%)	4 (36.4%)	4 (100%)	0 (0.0%)	0.241
Chest pain (yes)	2 (5.9%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	-
Haemoptysis (yes)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	-
Wheezes (yes)	2 (5.9%)	2 (18.2%)	0 (0.0%)	0 (0.0%)	0.365
Repeated Hospitalization (yes)	2 (5.9%)	0 (0.0%)	2 (50.0%)	0 (0.0%)	0.521
Relapse	1.21 ± 1.12	2.18 ± 1.78	2.25 ± 1.5	1.50 ± 0.71	0.125

Data are expressed as mean ± SD or number (%).

then cigarette and lastly mixed group. Actual FEV1/FVC more in cigarette group than hookah then other group and lastly mixed group. Both actual PEFR and TLC were more in hookah than others then cigarette then mixed group. Exhaled CO level was high in cigarette group followed by hookah then mixed and finally other group. No statistical significant differences were detected between groups regarding pulmonary functional parameters.

4. Discussion

Yet smoking still puts emphasis on more and more threats on human health so physicians especially pulmonologists still presage people about the medical as well as social benefits of quitting smoking whatever the degree of smoking and whatever the age of the quitter. This signifies not only the positive feedbacks of smoking cessation over and above giving the confidence of takeover this hardship with the smoking habit.

Clinical features detected in our subjects were harmonized and similar in both recent quitter subjects and remote quitters (Table 5). Clinical features showed higher percentage in remote quitters than recent quitters that owed to the prevailed anti-inflammatory impact of smoking on bronchial layer and muffling of features by ameliorated inflammatory cells during preceding smoking duration. Moreover there was no statistical significant difference in clinical findings between both recent and remote quitter subjects as in Table 3 including cough ($p = 0.688$), dyspnea ($p = 0.718$), chest pain ($p = 0.796$), wheezes ($p = 0.293$), hospitalization ($p = 0.634$) and relapse ($p = 0.159$).

All clinical features in Table 4 were higher in mixed group than other groups but without significant statistical differences also in Table 3. This finding despite not studied on large scale for smoking type however, this can be attributed to cumulative effects of smoking toxic metabolites on the long term time of smoking habit as well as synergistic impact of smoking harms on bronchial airways.

According to a study conducted by Willemse *et al.* [11], they studied the effects of smoking cessation on respiratory symptoms, lung function, airway hyperresponsiveness (AHR), and pathological and inflammatory changes. The

Table 5. Clinical data among classified groups.

	Recent quitter (n = 126)	Remote quitter (n = 180)	p value
Cough (yes)	60 (47.6%)	96 (53.3%)	0.688
Dyspnea (yes)	36 (28.6%)	60 (33.3%)	0.718
Chest pain (yes)	6 (4.8%)	6 (3.3%)	0.796
Haemoptysis (yes)	(0.0%)	0 (0.0%)	-
Wheezes (yes)	18 (14.3%)	6 (3.3%)	0.293
Repeated Hospitalization (yes)	6 (4.8%)	18 (10.0%)	0.634
Relapse	1.19 ± 1.03	1.73 ± 1.51	0.159

Data are expressed as mean ± SD or number (%). $p > 0.05$ = not significant.

reported prevalence of intermittent cough, expectoration and wheeze are 5 - 21, 5 - 30 and 1% - 19%, respectively, in ex-smokers, and 10% - 40% for both cough and expectoration and 7% - 32% for wheeze in smokers [12] [13] [14]. In contrast, the frequency of dyspnea is similar between ex-smokers and smokers (extending 2% - 41%), suggesting that the feeling of dyspnea is either not reversible after smoking cessation, or due to factors other than lung disease. In their study they did not classify ex-smokers according duration of cessation. Hence their study could give us idea about persistence of clinical symptomatology after smoking cessation.

Multiple cross-sectional researches propose that respiratory symptoms recover after smoking cessation. Nevertheless, these features seem not to disappear as the prevalence of respiratory symptoms in ex-smokers is described as higher than or similar to those established in nonsmokers [14] [15] [16] [17]. Longitudinal studies are in agreement with the above cross sectional studies, displaying that most intermittent symptoms decrease within 1 - 2 months after smoking cessation as that occurred with our recent subjects (recent quitter). The prevalence of cough and wheeze decreases to that in nonsmokers, whereas the prevalence of expectoration remains slightly higher [18] [19] [20]. Furthermore, symptoms are also less likely to develop later in life if smokers without chronic symptoms quit smoking. For instance, Krzyzanowski *et al.* presented that only 12% of quitters versus 29% of persistent smokers developed one of these symptoms [21] [22].

Unlike our study three studies, in which the duration of smoking cessation ranged 2 - 6 weeks to 1 - 12 yrs, showed no difference in the prevalence of dyspnea after smoking cessation [21] (**Table 6**).

Gaining body weight, as regularly occurs during smoking cessation, might explain the increase in dyspnea, but this was not inspected in this study. Paralleling to another study Peterson *et al.* [23] showed that dyspnea improved in 12 smokers after 1 and 18 months smoking cessation, despite an increase in body weight.

As declared above, dyspnea and chest discomfort in fit healthy smokers may be due to origins other than lung disease. In addition, it is unlikely that subjects

Table 6. Demographic data among classified groups.

	Recent quitter (n = 126)	**Remote quitter (n = 180)**	**p value**
Age (yrs.)	29.76 ± 7.63	30.83 ± 14.58	0.760
Duration of smoking (yrs.)	7.90 ± 4.44	7.77 ± 5.85	0.928
Duration of cessation (yrs.)	1.45 ± 0.57	7.48 ± 14.73	0.068
Occupation			
-Employee	60 (47.6%)	60 (33.3%)	0.304
-Students	66 (52.4%)	120 (66.7%)	

Data are expressed as mean ± SD or number (%). p > 0.05 = not significant.

without chronic respiratory symptoms experience the same degree of dyspnea as COPD patients. Furthermore, the cumulative or daily cigarette consumption is not always mentioned, and, if it is, also varies between the studies.

All pulmonary functional parameters illustrated showed higher levels and better measurement in remote quitter than recent quitter (Table 2) but without statistical significance differences between their mean values that made certain the matching and homogeneity between two groups regarding age and respiratory state.

There was statistical positive correlation (Table 7) between duration of smoking cessation and actual FEV1 ($r = 0.535$; $p = 0.702$), actual FVC ($r = 0.388$; $p = 0.299$), actual PEFR ($r = 0.415$; $p = 0.292$), actual TLC ($r = 0.548$; $p = 0.302$), functional defect FEV1 ($r = 0.624$; $p = 0.001$), functional defect of FVC ($r = -0.117$; $p = 0.461$), functional defect of PEFR ($r = 0.100$; $p = 0.517$) and functional defect of TLC ($r = -0.183$; $p = 0.257$) while statistical negative correlation between CO exhaled level ($r = -0.119$; $p = 0.406$).

Both actual FEV1, and FVC were more in group other followed by hookah then cigarette and lastly mixed group (Table 8). Actual FEV1/FVC more in cigarette group than hookah then other group and lastly mixed group. Both actual PEFR and TLC were more in hookah than others then cigarette then mixed group. Exhaled CO level was high in cigarette group followed by hookah then mixed and finally other group. No statistical significant differences were detected between groups regarding pulmonary functional parameters.

Other multiple cross-sectional studies have shown that FEV1 is lowest in individuals without chronic symptoms who smoke, highest in those who have never smoked and intermediate in ex-smokers [24] [25] [26]. One exception is the finding that ex-smokers aged > 70 years tend to have lower lung function than smokers of the same age. This finding can be ascribed to a "healthy smoker"

Table 7. Correlation between duration of smoking cessation and different pulmonary function parameters.

	Duration of smoking cessation	
	Pearson correlation	**p value**
Actual FEV1	0.535	0.702
Actual FVC	0.388	0.299
Actual PEFR	0.415	0.292
Actual TLC	0.548	0.302
CO exhaled level	−0.419	0.406
Functional defect of FEV1	0.624	0.001*
Functional defect of FVC	0.117	0.461
Functional defect of PEFR	0.100	0.517
Functional defect of TLC	0.183	0.257

$p > 0.05$ = not significant. *$p \leq 0.05$ = significant.

Table 8. Pulmonary functional data among patients classified according to type of smoking subgroups.

	Cigarette (n = 34)	Hookah (n = 11)	Mixed (n = 4)	Others (n = 2)	p value
Predicted FEV1	3.34 ± 0.06	3.40 ± 0.28	3.05 ± 0.17	3.70 ± 0.00	0.925
Actual FEV1	3.07 ± 0.49	3.23 ± 0.87	1.64 ± 0.07	3.26 ± 0.00	0.918
Functional defect of FEV1	0.91 ± 1.04	0.67 ± 0.31	1.41 ± 0.10	0.44 ± 0.00	0.832
Predicted FVC	4.24 ± 0.24	4.08 ± 0.22	3.66 ± 0.17	4.40 ± 0.00	0.854
Actual FVC	3.65 ±0.52	3.76 ± 0.81	2.53 ± 0.13	3.84 ± 0.00	0.833
Functional defect of FVC	0.70 ± 0.35	0.69 ± 0.26	1.14 ± 0.30	0.56 ± 0.00	0.854
Predicted FEV1/FVC	78.65 ± 4.12	82.82 ± 2.99	82.50 ± 0.58	96.00 ± 0.00	0.524
Actual FEV1/FVC	84.12 ± 8.70	83.45 ± 7.89	65.50 ± 6.35	74.00 ± 0.00	0.422
Functional defect of FEV1/FVC	0.07 ± 0.03	0.15 ± 0.00	0.17 ± 0.05	22 ± 0.00	0.325
Predicted PEFR	9.87 ± 0.47	9.89 ± 0.47	8.58 ± 0.49	10.12 ± 0.01	0.258
Actual PEFR	7.15 ± 2.38	8.65 ± 2.34	2.55 ± 0.06	8.20 ± 0.01	0.125
FFD of PEFR	3.12 ± 1.96	1.86 ± 2.38	6.02 ± 0.55	1.94 ± 0.01	0.152
Predicted TLC	5.34 ± 0.28	4.91 ± 0.78	4.78 ± 0.43	5.50 ± 0.00	0.825
Actual TLC	4.59 ± 0.62	4.83 ± 0.89	3.28 ± 0.03	4.80 ± 0.00	0.833
FFD of TLC	0.94 ± 0.54	0.82 ± 0.37	1.50 ± 0.46	0.70 ± 0.00	0.224
CO exhaled level	1.62 ± 0.85	1.55 ± 0.93	1.25 ± 0.50	1.00 ± 0.00	0.971
SpO_2	96.76 ± 1.07	96.64 ± 0.81	95.25 ± 1.50	97.00 ± 0.00	0.932

Data are expressed as mean ± SD.

effect [27] *i.e.* smokers who are not troubled by their habit continue to smoke (so-called healthy smokers), whereas smokers who are troubled by their habit are more likely to quit smoking. Most studies showed a substantial excess decline in FEV1 in smokers over nonsmokers, ex-smokers and quitters [28] [29] [30] [31].

Studies showed significant correspondence between them in the reported decline in FEV1 in smokers without chronic symptoms, ex-smokers, quitters and nonsmokers. Differences in age, baseline FEV1 or sex could not clarify to a major part this overlap although it may be attributed to differences in severity of bronchial hyperresponsiveness or prevalence of respiratory symptoms and [32] [33] [34].

In our study the duration of smoking and type of smoking played an important role in underlying this discrepancy. Our work denoted that all functional parameters were higher in mild than moderate than heavy quitter with significant statistical differences regarding FEV1 (predicted-actual and functional defect), actual PEFR, actual FVC. On another hand both actual FEV1, and FVC were more in group other followed by hookah then cigarette and lastly mixed group. Actual FEV1/FVC more in cigarette group than hookah then other group

and lastly mixed group. Both actual PEFR and TLC were more in hookah than others then cigarette then mixed group. Exhaled CO level was high in cigarette group followed by hookah then mixed and finally other group. No statistical significant differences were detected between groups regarding pulmonary functional parameters (Table 9).

Fletcher and PETO [35] put in their study inclusion of males with mild airway obstruction who showed that the accelerated decline in FEV1 in ex-smokers was slower than that in smokers (37 and 62 mL/yr^{-1}, respectively). In addition, POSTMA *et al.* [36] clarified that smoking cessation in smokers with moderate COPD reduced the accelerated decline in FEV1 by 50%, from 85 to 49 mL/yr^{-1}. The results in mild-to-moderate COPD patients of the Lung Health Study presented a parallel reduction in the 5 years subsequent to their date of smoking cessation, *i.e.* 63 mL/yr^{-1} in persistent smokers and 34 mL/yr^{-1} in quitters [37] [38] [39] [40]. After smoking cessation in the course of the first year, FEV1 upgraded by 57 mL in quitters, whereas it fell by 32 mL in persistent smokers [38], however when 11 years with follow-up performed, the decline in FEV1 in quitters was 30 mL/yr^{-1} for males and 22 mL/yr^{-1} for females, whereas, in continuous smokers, the decline was 66 mL/yr^{-1} and 54 mL/yr^{-1}, respectively [40]. The reported decline in FEV1 is intensely linked to cumulative cigarette intake and severity of

Table 9. Pulmonary functional data among patients classified according to smoking index subgroups.

	Mild quitter (n = 258)	Moderate quitter (n = 36)	Heavy quitter (n = 12)	p value
Predicted FEV1	3.36 ± 0.14	3.20 ± 0.09	2.90 ± 0.00	0.015
Actual FEV1	3.23 ± 0.52	1.93 ± 0.18	1.58 ± 0.00	0.006
Functional defect of FEV1	0.81 ± 1.00	1.27 ± 0.19	1.32 ± 0.00	0.008
Predicted FVC	4.20 ± 0.19	4.11 ± 0.53	3.52 ± 0.00	0.125
Actual FVC	3.74 ± 0.57	2.82 ± 0.32	2.64 ± 0.00	0.025
Functional defect of FVC	0.67 ± 0.32	1.08 ± 0.29	0.88 ± 0.00	0.241
Predicted FEV1/FVC	79.79 ± 3.66	78.00 ± 7.75	82.00 ± 0.00	0.231
Actual FEV1/FVC	86.19 ± 6.72	67.67 ± 3.14	60.00 ± 0.00	0.352
Functional defect of FEV1/FVC	0.08 ± 0.03	0.11 ± 0.05	0.22 ± 0.00	0.452
Predicted PEFR	9.89 ± 0.47	9.56 ± 0.46	8.15 ± 0.00	0.235
Actual PEFR	7.92 ± 2.09	3.12 ± 0.91	2.60 ± 0.00	0.041
FFD of PEFR	2.41 ± 1.72	6.44 ± 0.64	5.55 ± 0.00	0.052
Predicted TLC	5.30 ± 0.22	4.84 ± 1.20	4.40 ± 0.00	0.325
Actual TLC	4.71 ± 0.68	3.81 ± 0.48	3.30 ± 0.00	0.365
FFD of TLC	0.83 ± 0.41	2.02 ± 0.14	1.10 ± 0.00	0.918
CO exhaled level	1.58 ± 0.88	1.50 ± 0.55	1.00 ± 0.00	0.852
SpO_2	96.72 ± 1.03	96.83 ± 0.41	94.00 ± 0.00	0.933

Data are expressed as mean ± SD or number (%).

current bronchial hyperresponsiveness in smokers. Also connected to the number of cigarettes smoked: heavy smokers who had mild/moderate COPD revealed a more decline than light smokers, and these heavy smokers improved their FEV1 after smoking cessation than light smokers [41].

The decline in FEV1 return to normal age range 2 years after smoking cessation [42]. However, Xu, X., Weiss *et al.*, 1994 declared that a faster decline in FEV1 was found in ex-smokers than in nonsmokers (20 and 6 mL/yr^{-1}, respectively) but was equivalent in quitters as in nonsmokers in this study [31]. Only one longitudinal study, following a small number of cases, showed that FEV1 improved after smoking cessation [43] [44] but all other studies do not show this [41]. Already normal lung function in these participants before smoking cessation may be a probable explanation. Our subjects did not perform pulmonary function study as follow up assessment method to denote the rate of decline in FEV1 that was considered as one of the limitations that needs to be managed in more multicenter studies planned to be conducted in the future.

Several studies in smaller numbers of subjects have investigated the effects of smoking cessation on uneven ventilation and small airway closure using the single-breath nitrogen-washout test, however in our work no efforts was made to assess small air way function in outpatient clinic presented in N2 wash out test was conducted owing to compliance of cases in addition to necessity to admit these cases according Center of medical services in Taibah University hospital protocols more over small airway disease assessment need more than one test to be fully evaluated.

Rekha Chaudhuri, Eric Livingston, Alex concluded in their study that the mean (SD) change in FEV1 in ml in the quit group was 356 (278) at 1 week 390 (311) at 3 week and 450 (471) at 6 week of smoking cessation. There was no difference in the equivalent FEV1 measures in the smoking control group. In the comparison of quitters with control smokers at 6-wk cessation, there was a mean improvement of 407 ml in FEV1, 15.2% in FEV1% predicted and 93 L/min in PEF. They found that no correlation with FEV1 improvement and variables like age, sex, duration of smoking, atopy, pack-years smoked, dose of inhaled corticosteroids, or sputum cell counts [9].

However the decline in exhaled carbon monoxide in the quit group in Rekha Chaudhuri, *et al.*, study [9] compared with the control group at all patterns provided impartial proof for effective smoking cessation as minimum 24 to 48 h before each clinic evaluation.

In our work exhaled CO level was higher in remote group than those in recent group without statistical significant difference. Co level showed negative statistical correlation with duration of quitting smoking, it was higher in mild quitter than moderate quitter than heavy quitter without statistical significant difference. According type of smoking cigarette smoking demonstrated higher exhaled CO level in the four groups followed by hookah then mixed and lastly other group that may reflect the more toxic effect of smoking especially heavy smoker as well as duration of smoking. In our work Smoking cessation improves

respiratory symptoms and bronchial hyperresponsiveness, and prevents accelerated decline in lung function, in all smokers, with or without chronic obstructive pulmonary disease.

In chronic obstructive pulmonary disease patients, the established fibrosis and loss of alveolar connections is undoubtedly irreversible, enlightening why the FEV1 does not normalize after smoking cessation in them signifying positive impacts of smoking cessation on important inflammatory and/or remodeling processes in the lungs. Therefore, future research needs to investigate the relevant biological airway markers that identify these changes as well as pharmacological intervention [11]. Many limitations in our study was confronted presented in small number of studied cases as these type of studies needs much more cases secondly specification of age groups thirdly inclusion of females that was difficult of be considered and lastly sharing study with other universities at Al-Medina Al-Munwarah province.

5. Conclusion

Effective prevention of cigarette smoking and help for those wishing to quit can yield enormous health benefits for populations and individuals. Smoking cessation has been shown to be linked to prominent amelioration of respiratory symptoms in addition to significant improvement in pulmonary functional evaluation as well as exhaled CO level. Promoting and supporting smoking cessation programs should be an important health policy priority for healthcare professionals in all clinical settings.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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