Original Article

Risk Factors for Chronic Obstructive Airway Disease: A Hospital Based Prospective Study in Rural Central India

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Abstract

Background: Chronic obstructive pulmonary disease affected 215 million populations worldwide in 2016 and more than 90% deaths occur in low and middle-income countries. Only few studies have looked at this problem with rural perspective. This study aims to find prevalence of risk factors in COPD (chronic obstructive pulmonary disease) patients at tertiary care centre which mainly caters rural population. **Material and Methods:** This is a hospital-based longitudinal cohort study conducted at a rural based tertiary care Medical Institute in central India. Patient with history of cough and breathlessness for 3 months or more were studied. Spirometry-confirmed 113 COPD patients were studied for risk factors like male sex, advancing age, smoking, indoor air pollution and baseline assessment was done with Clinical COPD Questionnaire (CCQ). **Result:** Total 113 patients [median age, 59 y (interquartile range [IQR] 50-66); [82 (72.6% men and 31 (27.4%) women]) were included in study. Severity of COPD was not linearly correlated with the severity of risk factors like male sex (p=0.99), advancing age (p=0.70), smoking (p=0.78) and indoor air pollution (p=0.82). Low BMI (Body mass index) (18.1 Vs 20.1 Kg/m^2, p<0.01) was associated with severity of COPD. Women tended to spend more time in house (p<0.01) and Indoor risk time product grade was significantly higher in females (grade 4 indoor risk time product 30.9 vs 13.2, p=0.01). The CCQ score had an inverse relationship with baseline FEV1 (Forced expiratory volume in one second) [Pearson coefficient -0.33 (p=0.003)]. There was moderately strong relationship between decline in FEV1 and decline in PEFR (peak expiratory flow rate) (r=0.66). **Conclusion:** The CCQ may be used as low-cost and non-instrument-based indicator of FEV1 and PEFR to determine the severity of COPD at baseline as well as to judge improvement from baseline. CCQ score would be useful where spirometry is not available.

Keywords: COPD; Risk factors; Rural

Introduction

COPD (Chronic obstructive pulmonary disease) is leading cause of morbidity and mortality in countries of high, middle and low income. [1] The World Health Organization Global burden of Disease project [2,3] estimated that COPD was fifth leading cause of death in 2001 and will be third leading cause by 2020 and further increases in its prevalence and mortality can be predicted in the coming decades. [3] The impact of this respiratory disease worldwide is expected to increase with a heavy economic burden on individuals and society. [4]

COPD, hitherto underdiagnosed in India, is now recognized in 4-10% of adult male population of India and several other Asian countries. Almost all forms of smoking products such as cigarettes and bidis used were found to be significantly associated with COPD. In non-smokers, especially women, exposure to indoor air pollution from domestic combustion of solid fuels is an important factor. More significantly the exposure to environmental tobacco smoke was regarded an established cause for COPD. [5,6]

COPD is preventable and treatable disease with significant extrapulmonary effects that lead to comorbid conditions. [7] These include muscle wasting, cachexia, worsening comorbid diseases, such as ischaemic heart disease, heart failure, osteoporosis, normocytic anaemia, lung cancer, depression and diabetes. [8,9]

Only a few studies have looked at this problem with rural perspective. Targeting at modifiable risk factors will improve quality of life in COPD patients.

Materials and Methods

This is a prospective cohort study conducted at Kasturba Hospital Sevagram, a rural based tertiary care hospital. Patients who presented in the medicine wards or at the respiratory OPD with symptoms of production chronic cough with or without breathlessness lasting for more than 3 months were screened. Those with heart failure, parenchymal lung lesion on X-ray chest, renal failure, and other co-morbid conditions were excluded. The potential study participants, after their informed consents, underwent baseline assessment with Clinical COPD Questionnaire (CCQ) [10] and risk factor assessment along with clinical examination, X-ray chest and ECG. Pulmonary function test (by Spirolab III portable spirometer) was done to confirm COPD. This cohort was followed up at three months for a repeat pulmonary function testing.

How to Cite this Article: Soni NA, et al. Risk Factors for Chronic Obstructive Airway Disease: A Hospital Based Prospective Study in Rural Central India. Ann Med Health Sci Res. 2019;9:484-489

CCQ provides clinical status of airway, activity limitation of patient and emotional dysfunction. Severity of COPD can be measured by symptom score, functional score, and mental score with each question graded in 7-point scale. Mild and severe disease were defined by GOLD (Global Initiative for Chronic Obstructive Lung Disease) criteria. [11] Those in stage I and II were classified as mild disease and those in stage III and IV were as having severe disease. Frequency of smoking was estimated as occasional smoker (does not smoke every day but up to 6 per week), frequent smokers (smoke 1-2 cigarettes/bidi per day), regular smoker (smokes 3-6 cigarettes/bidi per day), moderate smoker (smokes up to 10 cigarettes/bidi per day), and heavy smoker (smokes >10 cigarettes/bidi per day). Risk grade for indoor air pollution is calculated based on self-reported presence of smoke in kitchen, use of wood / kerosene as fuel type, presence of mud walls and presence of thatched /tin roof. The responses from each of these components were added to obtain the "indoor exposure risk" ranging from 0-4. "Risk time product" was derived by multiplying Indoor exposure risk with average time spent indoor (determined from the questionnaire) as a cumulative measure of indoor air pollution risk. Risk-time product was graded 1 to 4 according to score (Grade 1: 0-3, Grade 2: 4-18, Grade 3: 19-36 and Grade 4: 37 or more).

Statistical analysis

We used Stata 11.0 (StataCorp. College Station, Texas, USA) to

Table 1: Demographic characteristics of study population.

Variables	Total	Patients with mild COPD GOLD I/II	Patients with severe COPD GOLD III/IV	p-value
Number	113	73	40	
Age (years)	57.6 (13.6)	57.6 (1.6)	58.6 (2.0)	0.70*
Gender				
Male	82	53 (64.6)	29 (64.5)	
Female	31	20 (35.4)	11 (35.5)	0.99†
Location				
Rural	88	54 (73.9)	34 (85.0)	
Urban	25	19 (26.1)	6 (15.0)	0.17†
Body mass index. median (IQR) (kg/m^2)	19.2 (4.1)	20.1 (4.2)	18.01 (3.5)	<0.01*
Cough duration, yr	6.2 (7.2)	5.9 (6.6)	6.6 (8.3)	0.63*
Breathlessness duration, yr	6.0 (6.8)	5.8 (6.4)	6.3 (7.8)	0.67*

Table 2: Prevalence of potential risk factors by severity of COPD.

Risk factors	Total	Patients with mild COPD GOLD I/II	Patients with severe COPD GOLD III/IV	p-value
Smoking History	59	37 (50.6)	22 (55.0)	0.66†
Pack-years of smoking				
Less than 10	13	8 (10.9)	5 (12.5)	
10 to 20	13	7 (9.5)	6 (15.0)	
20 or more	33	22 (30.2)	11 (27.5)	0.58‡
Type of fuel used				
Kerosene/Wood	62	37 (50.7)	25 (62.5)	
Electric /LPG	51	36 (49.3)	14 (37.5)	0.22†
Presence of smoke in kitchen	38	28 (38.4)	13 (32.5)	0.82†
Type of wall				
Mud	48 (42.5)	30 (41.1)	18 (45.0)	
Brick	30 (26.5)	19 (26.0)	11 (27.5)	
Plaster	35 (31.0)	24 (32.8)	11 (27.5)	0.83†
Type of roof				
Thatched	24	15 (20.6)	9 (22.5)	
Tin	17	11 (15.1)	6 (15.0)	
Kavelu	30	20 (27.4)	10 (25.0)	
Cement	42	27 (36.9)	15 (37.5)	0.99†

†Chi square test ‡ Mann Whitney rank sum test

Table 3: Gradation of indoor exposure risk in patients with COPD by gender.

Variables	All patients (n=113)	Male (n=82)	Female (n=31)	p-value
Smoking	59	59 (71.9)	0 (0)	<0.01
Time spend indoors				
<4 hrs	4	4 (4.8)	0 (0.0)	
4-8 hrs	26	23 (28.0)	3 (9.6)	
8-12 hrs	23	21 (25.6)	2 (6.4)	
>12 hrs	60	34 (41.6)	26 (84.0)	<0.01‡
Indoor risk-time product value	22.3 (20.8)	20.2 (18.0)	27.7 (26.1)	0.08*
Indoor risk-time product grade				
Grade 1, n (%)	26	18 (21.6)	11 (33.3)	
Grade 2, n (%)	36	32 (38.5)	4 (12.1)	
Grade 3, n (%)	30	22 (26.5)	8 (24.2)	
Grade 4, n (%)	21	11 (13.2)	10 (30.3)	0.01†

†Chi square test * student's t test

analyse data. We used student's two-tailed t test to compare means, Mann Whitney test to compare medians and $\chi 2$ or Fisher's exact test to compare proportions. All tests were two-sided with a 5% significance level. We analysed the relationship between the clinical COPD questionnaire and the spirometry values by using pairwise correlation. The Pearson correlation coefficient was used to denote the relationship. FEV1 (Forced expiratory volume in one second) is correlated with different components of the CCQ score (Symptom score, function score and mental score) and also with total score. For these correlations we used the CCQ score as a continuous measure, to compare with percentage of predicted FEV1 value. These correlations were performed for these two measurements taken at baseline and at three months follow up.

Results

Flow of patients in study has shown in Figure 1. Table 1 shows demographic characteristics of study population. Final dataset for study comprised of Total 113 patients [median age, 59 y (interquartile range [IQR] 50-66; range=27-85 years]; [82 (72.6% men and 31 (27.4%) women]). A little more than three forth (77.9%) of COPD patients in study belonged to rural areas. As compared to mild COPD, severe COPD patients tended to have a lower BMI (18.1 Vs 20.1 Kg/m^2, p<0.01). Mild and severe COPD patients did not differ in duration of symptoms like cough (p=0.63) or duration of breathlessness (p=0.67).

Table 2 shows prevalence of potential risk factors in mild and severe COPD patients. Mild and severe COPD patients did not differ in smoking intensity (pack-years of smoking) (p=0.66). Mild and severe COPD patients did not differ in regards to Indoor air pollution related risk factors such as cooking fuel (use of wood or kerosene as cooking fuel [p=0.22] and use of LPG or electric for cooking), presence of smoke in kitchen [p=0.82] (suggesting lack of ventilation) and poor housing structure [p=0.99] (house wall made up of mud, house roof thatched/tin).

No woman in our study sample ever smoked. Women tended to spend more time in house and were more likely to be exposed to indoor air

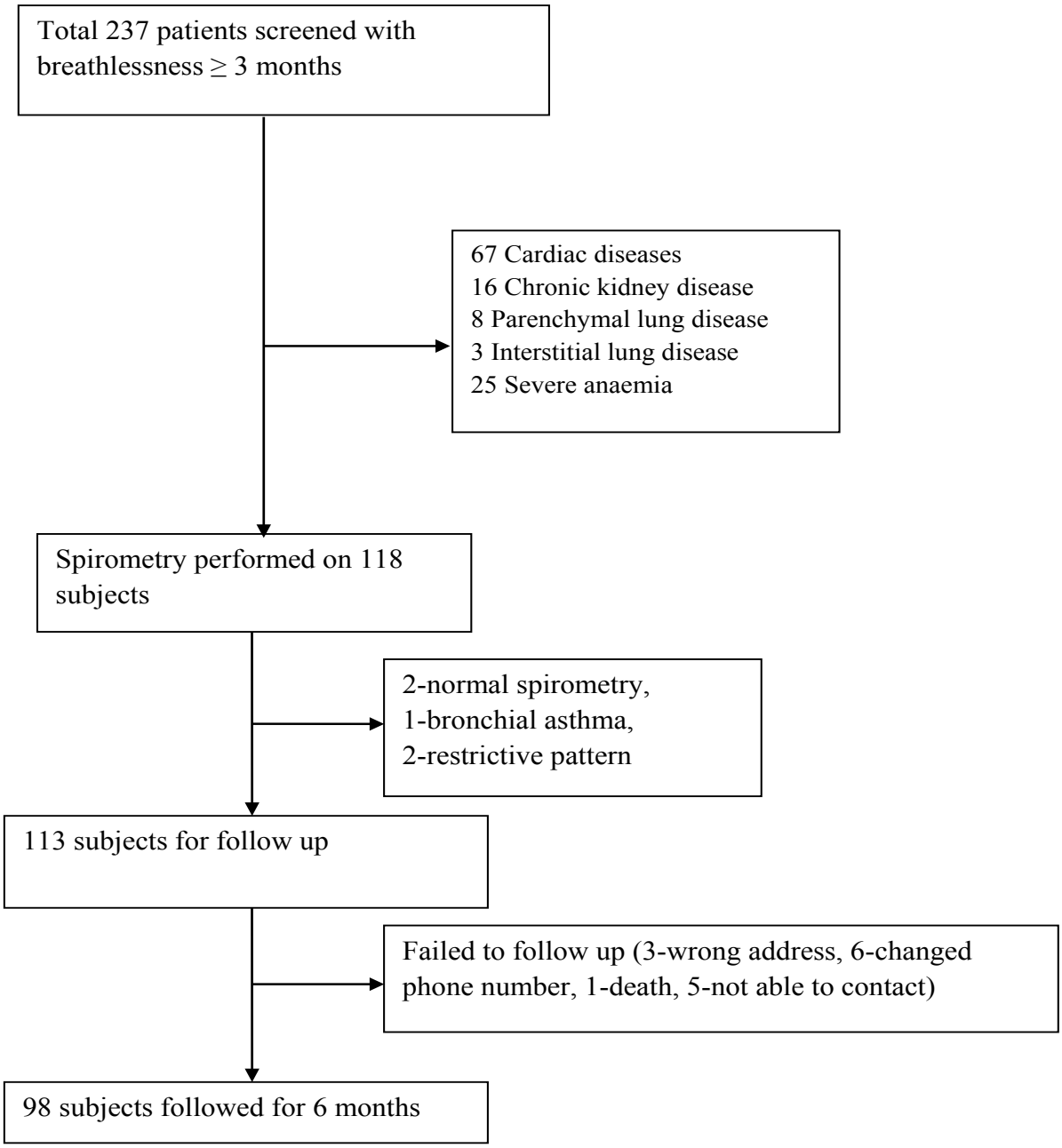
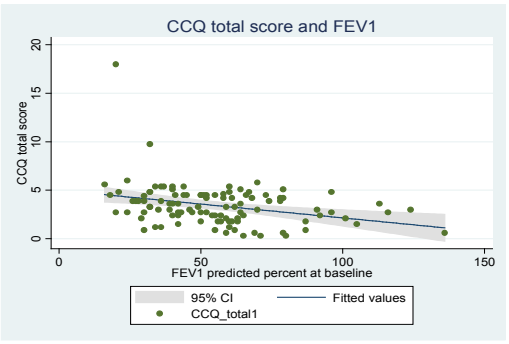
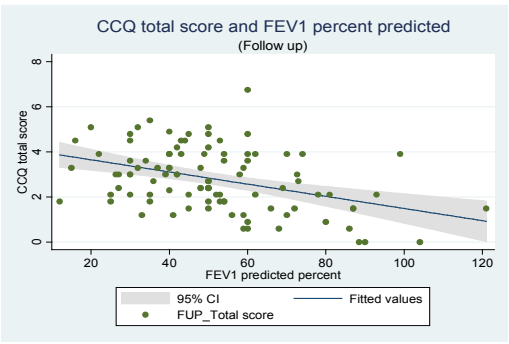


Figure 1: Study profile.

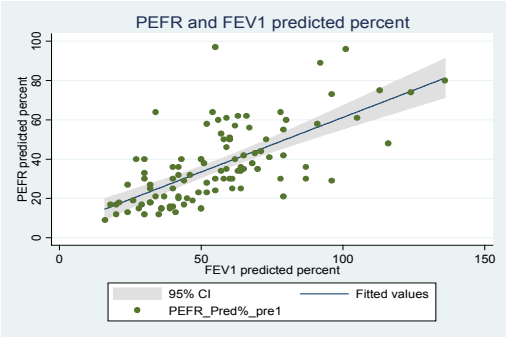


Pearson coefficient -0.33 (p=0.003) (baseline)

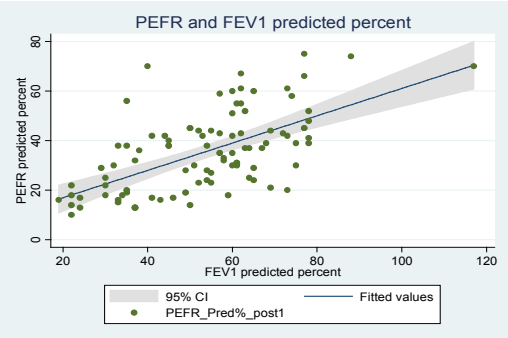


Pearson coefficient -0.43 (p<0.001) (follow up)

Figure 2: CCQ score at baseline and relation with FEV1 predicted percent at baseline and follow up.



Pre-bronchodialator Pearson coefficient 0.66 (p=<0.001)



Post-Bonchodialator Pearson coefficient 0.60 (p=<0.001)

Figure 3: The relation of spirometry indices FEV1 vs. PEFR at baseline.

pollution. The indoor risk-time product was 26.1% for females and 18% for males, indicating females were more at risk than males (grade 4 indoor risk time product 30.9 vs 13.2, p=0.01) [Table 3].

We found that as CCQ score increases, FEV1 declines. This indicates that CCQ has inverse correlation with FEV1. CCQ score was better correlated with FEV1 at baseline than at follow up [Figure 2]. Our data show that PEFR (Peak expiratory flow rate) and FEV1 are co-related (r=0.66). The r value indicates a moderately strong relationship. This relationship between PEFR and FEV1 persisted even after we measured the two variables after a bronchodilator, (r=0.60) [Figure 3].

Discussion

We found that prevalence of risk factors did not differ with severity of COPD. Severe COPD patients tended to have lower BMI. Females were at higher risk of developing COPD indicated by higher indoor risk-time product. CCQ had inverse correlation with FEV1 and CCQ score was better correlated with FEV1 at baseline than at follow up

Age

Most patients (95/113) in our study were aged 50 years or more which is comparable to Lange et al. [12] and Purohit et al. [13] studies showing high prevalence of chronic bronchitis in aged 65 years and 60 years or more respectively. A rise in prevalence with increasing age has also been reported by Ray et al. [14] Fletcher [15] and Holland. [16]

Sex

In the present study, 72.6% patients were male. Jindal et al. [17] and Studies around world [18] have observed a male preponderance in the prevalence of COPD. Of the several possible reasons which might account for a higher prevalence amongst males, is the difference in life style between males and female and may be genetic predisposition. Moreover, it is the habit of smoking which is common in males.

Smoking

Smoking as the cause of COPD has been irrefutably established through several major international reports [11,19,20] The same trend is visible in the Asian region. [6,21-25] Three fourth of the male (72%) patients were smokers in our study. The studies from North and South India [14,26-29] reported Smoker: non-smoker prevalence ratio among COPD patients which varied from 1.6 to 10.2:1. In our study we analysed smoker as a risk factor amongst male patient only as female patients included in the study had never smoked and in community also females were non-smoker. The Smoker: Non-smoker ratio among male was 2.56:1.

Relationship between severity of smoking and severity of COPD

The longitudinal studies [24] have shown rapid fall in the FEV1 in a smoking dose-response relationship i.e., larger the frequency and duration of smoking, the more the chances that one develops COPD. In natural History of COPD, the mean values of FEV1 decline in healthy men and women are around 30 ml/yr and 25 ml/yr respectively. Adult smokers experience on an average an FEV1 decline of 40-50 ml/yr, an excess of the normal annual decline of 15-20 ml/yr. In addition to being faster than normal, the decline of FEV1 may start earlier than usual. It seems that in smokers the plateau phase may be shorter or missing. This result in an earlier onset of decline in lung function which may contribute to development of COPD. In our study 50.6% of patients of

mild COPD patients and nearly half of those (55%) of severe COPD were smokers. 30.2% of mild COPD and 27.5% of severe COPD had history of more than 20 pack years of smoking. In our study no relationship was observed between smoking dose in terms of pack years and the severity of COPD. Actually, number of pack years only may not represent the smoking dose, as certain other factors like duration of each puff, ventilation of place of smoking, cigarette or bidi used, force of inhalation, size of butt etc are likely to affect the actual dose. However, these factors are not taken into consideration while analyzing this relationship. This may be the reason that relationship between smoking dose (calculated by pack years only) and severity of COPD was not observed. In the available literature studies on such relationship could not be traced.

Indoor air pollution: Other risk factors for COPD are indoor air pollution, environmental tobacco smoke exposure, lower socioeconomic status and rural residence. Jindal et al. argued that exposure to biomass fuel combustion could explain the high prevalence of COPD in rural women. [5,30] In addition to using biogas (like wood o cow dung) to cook food, poorly ventilated houses also increase the risk of COPD especially among women in developing countries. [31-34] In the present study half of our study patients (54%) use smoke producing fuel. Half of women (48%) lived in houses where smoke was likely to be trapped and 84% of the women sampled said that they tended to spend most of their time (more than 12 hours) at home. Thus, exposing women to smoke all through their life. The indoor risk- time product was 26.1% for females and 18% for males indicating that females were more at risk of indoor air pollution (p=0.08). The grade 4 indoor risk- time product value was 30.3% for females and 13.2% for males (p=0.01). Dusty environment of poor housing condition like mud walls, thatched roof, moreover small crowded rooms with smoke trapped within make person more prone to lung infections. The women and elderly who spent most of the time at home are more exposed population than working men. Our study shows that although women in rural areas do not smoke rather they are exposed to indoor air pollution which might increase their risk of developing COPD.

Body Mass Index: Patients with COPD have a 1 in 4 chance that they will develop significant malnutrition. (Fat-free body mass index <17 kg/m^2 (males) or <14 kg/m^2 (females)). [35] We found that patients with severe COPD tended to have a lower BMI (18.1 vs. 20.1 kg/m^2) compared to those with mild COPD. Patients with lower BMI also tended to have poorer FEV1. Advanced COPD is often accompanied by systemic wasting, with significant weight loss, bitemporal wasting, and diffuse loss of subcutaneous adipose tissue. Such wasting is an independent poor prognostic factor in COPD. According to Celli et al. [36] BMI <=21 is associated with poor prognosis in patients with COPD. In the present study, BMI differed significantly between groups of COPD patients. Most patients were severely malnourished: 79 patients had BMI <21 and 52 patients had BMI <18. In fact, 13 patients had BMI <15: Suggestive of severe adult malnutrition. The relation between BMI and CCQ scores in our COPD population was non-linear since scores tend to be worse with both decreasing BMI values below 15 and increasing values above 21.

Our study has several strengths. We enrolled a consecutive series of inpatients that fulfilled our inclusion criteria and ensured that all screened patients received spirometry. We applied American Thoracic criteria to judge the quality and appropriateness of the spirogram. Only a few patients were lost to follow-up.

We might have underestimated the prevalence of smoking in our study population as patients often hide smoking history because it is taboo in our culture. In some patients it was difficult to obtain an accurate and reproducible spirogram some did not understand our instructions or failed hard and long enough to generate a spirogram we could trust.

Conclusion

In summary, important risk factors of COPD such as Male sex, advancing age, smoking and indoor air pollution did not differ with severity of COPD. Severe COPD patients had low BMI. CCQ score had an inverse relationship with baseline spirometric values FEV1. Decline in FEV1 and PEFR were linearly corelated. CCQ score may be used as low lost, non-instrument-based indicator of FEV1 and PEFR to determine severity of COPD at baseline as well as to judge change from baseline.

Conflict of Interest

The authors disclose that they have no conflicts of interest.

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