Preserved ratio impaired spirometry: clinical, imaging and artificial intelligence perspective

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Abstract: Preserved ratio impaired spirometry (PRISm) is a pulmonary function pattern characterized by a forced expiratory volume in one second (FEV1) to forced vital capacity ratio greater than 0.70, with an FEV1 that is below 80% of the predicted value, even after the use of bronchodilators. PRISm is considered a form of "Pre-Chronic Obstructive Pulmonary Disease (Pre-COPD)" within the broader scope of COPD. Clinically, it presents with respiratory symptoms and is more commonly observed in individuals with high body mass index, females, and those who are current smokers. Additionally, it is frequently associated with metabolic disorders and cardiovascular diseases. Regarding prognosis, PRISm shows considerable variation, ranging from improvement in lung function to the development of COPD. In this article, we review the epidemiology, comorbidities, and clinical outcomes of PRISm, with a particular emphasis on the crucial role of imaging assessments, especially computed tomography scans and magnetic resonance imaging (MRI) technology, in diagnosing, evaluating, and predicting the prognosis of PRISm. Comprehensive imaging provides a quantitative evaluation of lung volume, density, airways, and vasculature, while MRI technology can directly quantify ventilation function and pulmonary blood flow. We also emphasize the future potential of X-ray technology in this field. Moreover, the article discusses the application of artificial intelligence, including its role in predicting PRISm subtypes and modeling ventilation function.

Keywords: Preserved ratio impaired spirometry (PRISm); chronic obstructive pulmonary disease (COPD); computed tomography imaging (CT imaging); magnetic resonance imaging (MRI)

Introduction

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines defined an unclassified GOLD type known as "Preserved Ratio Impaired Spirometry (PRISm)" a decade ago. Based on the GOLD 2025 report, "Pre-Chronic Obstructive Pulmonary Disease (Pre-COPD)" is identified in individuals who exhibit respiratory symptoms and/or structural lung abnormalities and/or physiological irregularities without the presence of airflow obstruction, as indicated by a forced expiratory volume in one second (FEV1) to forced vital capacity (FVC) ratio exceeding 0.70 in post-inhalation bronchial spirometry. The term "PRISm", recognized as a subtype of Pre-COPD, is characterized by an FEV1/FVC ratio of ≥0.7 post-bronchodilation, along with an FEV1 that is less than 80% of the predicted value [1-3]. This term is primarily applied in clinical contexts where individuals are at risk for developing COPD, such

as smokers, those with a history of prematurity, individuals with alpha-1-antitrypsin (AAT) deficiency and others who meet the criteria for Pre-COPD. This classification arises due to a deficit in both the FEV1 and FVC, which results in a maintained FEV1 to FVC ratio (4) with the potential to evolve into COPD (5,6). Certain individuals with PRISm may present with restrictive ventilatory abnormalities, marked by an FEV1/FVC ratio ≥0.7, FEV1 less than 80% of the predicted value, and FVC less than 80% of the predicted value, a condition referred to as "restrictive PRISm" or "restrictive spirometric pattern" (7,8). However, this pattern is associated with restrictive diseases and shows FVC and total lung capacity (TLC) decreased, but in obstruction diseases like COPD, TLC should be normal or increased estimated by body plethysmography (9,10). Therefore, such "PRISm-like restrictive pattern" is different in the clinical contexts. In addition, some studies have shown that the prevalence of PRISm using pre-bronchodilator spirometry is similar to the incidence of PRISm reported using postbronchodilator spirometry (3,11,12). Till now, several studies already performed spirometry without bronchodilator data for further research (3,4,13-16).

Patients with PRISm and COPD may both have similar symptoms such as dyspnea, chronic cough, and increased sputum, which may lead to misdiagnosis of PRISm as COPD in the absence of detailed pulmonary function test (PFT) results (3,17), and half of the PRISm population are asymptomatic (18). Therefore, understanding the association between this pattern of non-obstructive lung function abnormalities, and adverse clinical outcomes is significant for understanding the diversity and complexity of chronic lung disease.

Epidemiology of PRISm

The prevalence of PRISm has been estimated to vary between 3–20% (2,5,19-21), and previous research has indicated that the occurrence and outcomes of PRISm vary based on geographical location, as well as racial and ethnic factors (1,22) except for US cohort (3). PRISm has strong connection with a high body mass index (BMI) (3,5,23-25), underweight (3), female (3,24,25), current smoking (3,4,26), asthma (4), restrictive lung disease (12), air pollution (3), and heavy metals (27). Zhang *et al.* (28) found that nasal allergy symptoms also pose a risk for PRISm, and patients with obstructive sleep apnea (OSA) are 1.883 times more likely to develop PRISm than non-OSA patients. Moreover, the role of abnormal pulmonary growth and development

as an underappreciated risk factor for PRISm is not yet fully recognized. Several researchers have identified that impaired growth or nutritional deficiencies during fetal development (29), lung infections in childhood (30), and air pollution may contribute to the emergence of restrictive spirometric patterns in adulthood. It has been reported that patients with PRISm may progress to COPD varying between 15–49.4% (5,21). Additionally, 15.7% of PRISm cases may experience reversal (5). Therefore, early identification of PRISm, especially the subgroup that progresses to COPD, is crucial.

Co-morbidity of PRISm

The latest genome-wide association study on PRISm (31) indicates a relationship between PRISm and metabolic and cardiovascular diseases. It found that 18 out of 22 genetic signals correlated with traits related to diabetes, and seven were associated with variations in blood pressure. These findings may help to elucidate why PRISm is consistently linked to metabolic and cardiovascular diseases.

In 2016, a study analyzed the relationship between lung function and the onset of diabetes in 7,080 participants of the COPDGene study and found that PRISm was associated with the onset of diabetes (32). Furthermore, among PRISm participants with a history of type 2 diabetes (T2D), longer disease duration, greater use of glucose-lowering medications, and poorer disease control (indicated by higher hemoglobin A1c values) were reported (6), which may even lead to diabetic kidney disease and diabetic retinopathy (6). Based on the literature, the coexistence of PRISm with metabolic disorders is more probable than its association with COPD (26,33). Meanwhile, persistent PRISm findings and airflow obstruction contribute to a higher incidence of major adverse cardiovascular events (MACEs) (19,34-36), especially stroke, myocardial infarction, unstable angina and congestive heart failure (6). While the people who turn PRISm to normal spirometry (NS) findings would have a lower risk of MACE (26). According to the American cohort study involving 53,701 participants, individuals who have cardiovascular disease with PRISm exhibit a notably higher mortality rate than those with normal lung function or in the COPD group (3). The research of Li *et al.* (37) also confirmed this. PRISm's strong link to cardiovascular and metabolic diseases, exceeding its ties to COPD, likely results from increased BMI and hypoxia due to decreased FVC and FEV1. The resulting tissue hypoxia may trigger inflammation, leading to vascular complications

through cytokine release and immune cell activation (38). Another comorbidity associated with PRISm is lung cancer. Apart from smoking status, both moderate to severe COPD (GOLD 2–4) and PRISm are linked to an increased risk of lung cancer (39). PRISm is less common in adenocarcinomas and more common in squamous cell or small cell tumors in patients with lung cancer (39). The reason for this association needs further exploration, and the link between PRISm and lung cancer warrants further investigation.

Imaging assessment of PRISm

Although PFT is the gold standard for the diagnosis of PRISm, they only reflect the whole functional status of the lungs and may not detect focal morphological abnormalities that can only be identified through other diagnostic procedures, such as imaging (40). In patients at high risk for smoking, computed tomography (CT) detected disease progression may precede significant changes in lung function (41). The COPDGene study found that individuals with normal PFTs, including those with PRISm, could exhibit radiological abnormalities (42). This subset of people with radiological abnormalities is at a high risk of developing COPD during follow-up, highlighting the potential of imaging in the assessment of PRISm. Current imaging assessments mainly focus on CT quantification, magnetic resonance imaging (MRI) functionality and X-ray, while the advancement of artificial intelligence (AI) offers new avenues for exploration.

Qualitative evaluation of PRISm

Subjects with PRISm might show emphysema on chest CT. Radiologists can assess the distribution of emphysema directly on CT. Different distributions of emphysema were found to exist for PRISm and COPD on chest CT, the emphysema distribution of the left lung in the early COPD group covered more of the lower lobe, while in the PRISm the distribution was shifted toward the upper lobe (43). The Fleischner Society guidelines classify emphysema into three main types: central lobular emphysema (CLE), panlobular emphysema, and paraseptal emphysema. CLE is further divided into mild CLE, moderate CLE, confluent CLE, and progressive-destructive types. The relationship between PRISm and these categories of emphysema requires further investigation to determine if a closer association exists with any particular form.

Quantitative evaluation of PRISm

Concurrently, CT scans enable quantitative evaluation of PRISm. The quantitative assessment of PRISm predominantly focuses on lung volume, lung density, airway, and small pulmonary blood vessels. This process yields comprehensive anatomical and quantitative data.

Lung volume quantification

CT lung volume measurement involves scanning the entire lungs during inspiration or paired respiration. Using post-processing software, the lung tissue is separated from other chest structures to create a 3D model, and lung volume metrics like TLC, residual volume (RV) and functional residual capacity (FRC) are then quantified (44). Previous studies have illustrated an excellent correlation between CT-derived estimates of TLC and measurements obtained from helium dilution and body plethysmography (45), which is regarded as more sensitive than pulmonary function measurement (46). The article of Arjomandi et al. (47) have shown that for people with normal lung function (>0.7), those with higher ratios of CT-measured RV and TLC are more likely to develop COPD.

In a longitudinal study of lung function cohorts, Wan et al. (21) implicated that individuals with PRISm and higher predicted TLC and more air trapping were more likely to develop significant airflow obstruction (classified as GOLD stages 1 to 4) over time. These individuals also faced a higher risk of death. In contrast, those with stable PRISm throughout the study showed lung structures on CT scans that were similar to those with stable normal lung function (GOLD 0) (21).

Lung density quantification
Emphysema
The percentage of pixels in the low-density areas, characterized by an attenuation value below –950 Hounsfield units (HU), on the CT images obtained during the inspiration phase, is termed as the low-attenuation area percentage at –950 HU (LAA%), which is also recognized as the emphysema index (EI), calculated by the formula: EI = total emphysema volume (TEV)/total lung volume (TLV) (41,48). Also, there have been studies indicating that a negative relationship exists between EI and FEV1/FVC (49-51), while a decline in FEV1 is linked to emphysema progression in PRISm, this connection is not robust (41,52). Scholars postulate that PRISm patients with higher percentages of emphysema and air trapping also had higher mortality rates (21,53).

Air trapping

Air trapping which refers to an excessive amount of air remaining in the lungs after a normal breath is exhaled, is a sign of obstructive pulmonary conditions, it is associated with lower rates of exercise capacity and respiratory symptoms (48). This condition is observable on CT scans at the end of expiration, appearing as areas of pulmonary tissue that exhibit a reduction in attenuation and a failure to decrease in volume as expected. Utilizing both inspiratory and expiratory phases allows for the acquisition of additional parameters. These include the ratio of mean lung density between expiration and inspiration (E/I MLD), the ratio of FRC to TLC (33,41), the air trapping index (ATI) (54), the relative volume change within the attenuation range of –856 to –950 HU (RVC856–950), the expiratory attenuation at –856 HU (Exp-856) (54), and the parametric response mapping (PRM). Analysis through PRM can be implemented to assess the status of small airway diseases with functional implications. The pertinent parameters encompass the volume and percentage measurements of PRM-defined normal tissue (PRMnormal), PRM-identified emphysema (PRMemphysema), and PRM-determined functional small airway disease (PRMfSAD) (55,56). The decrease in FEV1 accounts for less than 10% of the progression of emphysema and less than 50% of air trapping detected by CT (41). Young *et al.* (36) studied airway dominant and emphysema dominant CT manifestations in over 4,000 patients in the COPDGene® cohort who underwent CT scans at baseline and 5 years later. The study conversion from PRISm to GOLD2–4 (over 30%) occurred with both CT patterns. Therefore, compared to the changes in FEV1, quantitative CT parameters allow researchers to monitor subtle changes in emphysema. These parameters can more directly reflect the physical changes in lung tissue, rather than being based solely on functional measurements of airflow limitation. This suggests that PRISm airflow obstruction assessed by CT is more informative.

Airway quantification

Quantification evaluation of the airways begins with the segmentation of the tracheobronchial tree during the inspiratory phase, extending at least to the sixth level of the bronchial tree. This process is followed by the evaluation of quantitative airway parameters, which include the thickness of the bronchial walls, the cross-sectional area of the bronchial walls, the area of the bronchial lumen, the diameter of the bronchi, and the area of the bronchiolar lumen. Additionally, assessments are made of the bronchial lumen diameter, the percentage of the bronchial wall area (%WA), and the squared tube diameter (Pi10), which is calculated for a diameter circumference of 10 mm which can assess small airway obstruction and explain the presence of respiratory symptoms beyond the information provided by PFTs (57,58).

Radiologic studies have revealed an increase in the percentage of wall area in male subjects with COPD. A study has compared the lung function of normal individuals with those suffering from PRISm and found that a higher subsegmental airway wall area is a significant influencing factor (1). Concurrently, they conducted univariate and multivariate regression analyses to examine the relationship between the small airway wall area and PRISm. The results indicated that this correlation remains significantly consistent regardless of the presence or absence of a bronchodilator response or chronic bronchitis (1). An analysis of Pi10 in individuals without airflow limitation (FEV1/FVC >0.7) revealed that those with higher Pi10 values experienced a faster annual decline in FEV1 (9% per year) and a higher incidence of COPD (2.22 times per standard deviation) (59). Hence, Labaki *et al.* (60) conducted research on 4,387 individuals with NS, 1,262 with PRISm, 2,713 with GOLD stages 1–2, and 1,770 with GOLD stages 3–4. The average Pi10 was 3.68 mm, the lowest in the NS group (3.65 mm) and the highest in the PRISm and GOLD 1–2 groups (3.73 and 3.75 mm, respectively). Additionally, Pi10 was significantly associated with higher all-cause mortality in the PRISm and GOLD 3–4 groups (60). At the same time, Pi10 is correlated with decreased FEV1 and can also predict decreased lung function (61).

Wei *et al.* (43) compared airway wall area, EI, and lung capacity in three groups: 80 patients with chronic bronchitis and normal lung function, 80 patients with chronic bronchitis exhibiting PRISm, and 187 patients with mild to moderate stage of COPD (FEV1 >50%). The results revealed that, compared to the chronic bronchitis group with normal lung function, the PRISm group had an elevated WA%LUL5 (percentage of the wall area of the fifth-generation bronchioles in the left upper lobe of the lung), reduced lung volumes, and increased mean lung density (43).

Pulmonary vascular quantification

Pulmonary artery enlargement (PAE) is used as a surrogate for pulmonary vascular disease and is associated with severe acute exacerbations and all-cause mortality in patients

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with PRISm (62). Chest CT can be used to assess PA by measuring the diameter of the PAE (62). Also, CT images provide high-resolution visualization of the small blood vessels in the lungs. Quantitative analysis software facilitates the measurement of the cross-sectional area (CSA) of these vessels and realizes the calculation of the total number and the density of them within a given surface area of the lungs. This allows for the evaluation of the size, morphology, and number of blood vessels. It also aids in identifying abnormal vessels, such as those exhibiting changes in morphology like twisting, dilation, or constriction, which are indicative of conditions like PRISm and vascular remodeling. Chinese researchers (63) analyzed quantitative CT parameters, including PRM, airway, and vascular parameters, in patients with PRISm. The ultimate findings revealed that these quantitative parameters could differentiate between PRISm, normal subjects, and patients with mild COPD. For the PRISm group, there were significant differences in vascular damage compared to subjects with normal pulmonary function, leaning more towards mild to moderate COPD.

MRI and X-ray assessment of PRISm

Not only CT, but MRI technology also holds significance for assessing ventilation function in PRISm. Through conventional ^{1}H MRI or direct imaging of exogenous gases using multinuclear MRI and spectroscopy, we can directly quantify ventilation, identify disease progression, directly measure airspace dimensions, and quantify pulmonary perfusion (64). Researches found that the barrier uptake, erythrocyte transfer, and erythrocyte/barrier ratio derived from ^{129}Xe MRI correlated well with the diffusing capacity for carbon monoxide (DLCO), enabling a better assessment of pulmonary air-blood exchange function (65). Additionally, it can detect subtle changes in ventilation and is more sensitive than FEV1 (65,66), which is beneficial for identifying populations that respond more to bronchodilators. Patients with COPD typically exhibit pulmonary perfusion defects (65) and the pulmonary perfusion defects in PRISm require further investigation. Moreover, some investigators have used MRI scans of the brain finding that lower pulmonary function parameters (e.g., FEV1% predicted and FVC% predicted) are associated with greater white matter lesion volume (35). Hence, the application of MRI in PRISm is noteworthy.

X-ray is not widely used in the field of Pre-COPD. Nevertheless, X-ray, with its low radiation, cost-effectiveness, and ease of operation, will play a potential role in the early screening of Pre-COPD patients. Willer et al.'s (67) research indicated that X-ray dark-field chest imaging technology had great potential in diagnosing COPD-related emphysema, showing a higher correlation in assessing lung diffusion capacity compared to CT-based parameters. Accordingly, this technology is expected to become a low-radiation alternative to CT scans for the diagnosis of COPD. Furthermore, Japanese scholars' research on dynamic digital radiography (DR) has revealed its broad prospects in COPD research. Dynamic DR can monitor changes in lung area and tracheal diameter during breathing, measure the rate of tracheal stenosis, and assess the movement of the diaphragm, all of which are closely related to the ventilation impairment in COPD patients (68,69). Therefore, X-rays might show great potential in evaluating the progression of PRISm to COPD.

Imaging-based AI for PRISm

AI has made significant progress in multiple fields. Currently, the application of AI in lung imaging is primarily focused on chest CT and X-rays. Deep learning is transforming the segmentation and quantification of lung structural components, such as using the U-net architecture for the segmentation of the lungs and lobes (70), and evaluating ventilation imaging through deformable image registration (DIR) (71). Additionally, radiomics can precisely identify COPD (72) and its co-morbidities (73). At the same time, AI also holds great significance in the typing of PRISm. Utilizing an unsupervised k-means cluster analysis with six key variables, including TLCCT as a percentage of predicted value, FEV1%, FEV1/FVC ratio, percent emphysema (%LAA-950insp), BMI, and segmental wall area percentage in three distinct subgroups (a restrictive subgroup, a COPD subgroup, and a metabolic subtype among patients with PRISm) were identified (53). These different subtypes may be associated with varying pathophysiological mechanisms. CT imaging was instrumental in identifying these subtypes, enhancing our understanding of PRISm's heterogeneity. A study develops a deep learning algorithm to predict pulmonary function from low-dose CT images, showing high accuracy in classifying respiratory high-risk groups (74). This model can predict specific FVC and FEV1 values for each individual from chest CT scans, thereby distinguishing high-risk populations, including PRISm. Also, Park et al.'s (75) research proved the application of deep learning enables the quantification of pulmonary emphysema on CT scans,

Table 1 Predictive factors for the progression of PRISm to COPD

Risk factors	References
Increased CT-measured RV/TLC ratio	(47)
Increased EI (LAA% on CT)	(41,48)
Higher predicted TLC	(21)
More air-trapping	(21)
Increased Pi10	(57)
Greater bronchodilator response	(80)
Chronic bronchitis with SAD	(15)
Consistent PRISm	(23)

PRISm, preserved ratio impaired spirometry; COPD, chronic obstructive pulmonary disease; CT, computed tomography; RV, residual volume; TLC, total lung capacity; EI, emphysema index (total emphysema volume/total lung volume); LAA, low-attenuation area percentage at −950 Hounsfield units; Pi10, square root of the wall area of an airway with a 10 mm internal perimeter; SAD, small airway disease.

presenting itself as a promising predictive instrument for assessing the risk of long-term nonaccidental mortality in asymptomatic individuals. In the future, we may be able to differentiate between high- and low-risk PRISm patients through AI, resulting in better management of PRISm patients.

Clinic outcomes and treatment

Previous studies demonstrated that the PRISm pattern has worse survival trajectories than mild COPD (19) and normal lung function (16,17,76). In contrast to normal lung function, PRISm increases all-cause mortality, respiratory-related mortality rates and cardiovascular-related mortality (3,5,16,77,78). Even compared to patients with COPD GOLD 2–4, PRISm patients' all-cause mortality is partly higher (5,21,79). One study proposed that if the PRISm condition is addressed early on, past episodes of PRISm may not impact long-term health outcomes if they are resolved in early adulthood (16). This hints that early detection, as well as early intervention, has a positive prognosis for PRISm.

PRISm can exhibit variability over time, with some individuals experiencing an escalation in airflow limitation, potentially advancing to COPD, while others either maintain their PRISm status or return to a normative spirometric configuration (5,6,80). Indicators of the

transformation of PRISm into COPD include persistent PRISm, small airway obstruction, a greater response to bronchodilators, and a decline in DLCO. It has been found that during follow-up, for those individuals with multiple confirmations of PRISm, the likelihood of developing COPD is five times higher than those with a single-time diagnosis of PRISm and the normal population even in non-smokers (23). Some researchers found patients with PRISm are often associated with small airway obstruction. A cross-sectional study based on a Chinese community showed that SAD parameters [two of pulmonary function, maximum mid-expiratory flow (MMEF), forced expiratory flow at 50% of forced vital capacity (FEF50), and forced expiratory flow at 75% of forced vital capacity (FEF75) <65%] were significantly reduced in patients with PRISm, even after the use of bronchodilators (33). Meanwhile, the researchers measured the impulse oscillometry parameter [a more accurate predictor of SAD (81)] and confirmed SAD was also significantly increased in patients with PRISm. To further investigate whether PRISm patients with small airway obstruction are more likely to develop COPD, Fan *et al.* (15) used pre-bronchodilator (pre-B) lung function data to observe changes in patients with PRISm who developed COPD and explored the relation of SAD and PRISm whose final results suggest that PRISm is an independent prognostic factor for COPD only when chronic bronchitis and SAD/MEEF are both diminished. Woodruff *et al.* (82) mentioned a greater airway response to bronchodilators was demonstrated for smokers with FEV1/FVC >0.7 who had a proportionately higher increase in FVC after inhaling bronchodilator, which is more likely to evolve COPD (1). A longitudinal study of PRISm measured PRISm's single DLCO, found the overall survival was significantly lower in the low DLCO group than in the preserved DLCO group (83). They found subjects who developed from NS to PRISm experienced the most significant decline in FVC, while those who transitioned from PRISm to COPD exhibited a less pronounced decline in FVC but a more severe decrease in FEV1. FVC decline in PRISm is influenced by different factors than in COPD (5). For individuals of PRISm who are more likely to develop COPD, we have compiled a list of factors that have been confirmed by researches (*Table 1*). Additionally, there are several potential predictive factors that may serve as biomarkers for the progression of PRISm to COPD.

Patients with PRISm can develop frailty, but there is still lacking explanation. He *et al.* (13) convey that the annual added value of the frailty index (FI) is highest in those with

consistent PRISm (0.301), followed by those who transition from NS to PRISm (0.242), and then those with COPD (0.172). Additionally, individuals who transitioned from PRISm to NS did not show accelerated FI progression compared to those who maintained NS, suggesting that a reversal from PRISm findings to NS may delay the progression of frailty. This is in line with previous study showing that the former history of PRISm does not appear to affect long-term morbidity and mortality if lung function is restored in early adulthood (16).

There is no clear treatment strategy for PRISm, earlier research has shown that inhaled dual bronchodilator treatment did not reduce respiratory symptoms in a tobacco-exposed population with normal lung function, and the treatment group with inhaled bronchodilators did not show a significant increase in lung function, with a mean change in inspiratory capacity of only 0.12 L compared with 0.02 L in the placebo group (84). The 2023 edition GOLD recommends that patients with PRISm should receive the following treatment and follow-up: first, quit smoking and undergo regular medical follow-ups, which include periodic BMI assessments and PFTs. Concurrently, routine chest CT scans should be conducted to monitor changes in lung structure and potential complications. Control of pulmonary infections and engagement in pulmonary rehabilitation training, such as strengthening the strength and endurance of respiratory muscles, are also essential. Appropriate supplementation with exogenous AAT and human recombinant surfactant proteins (SPs) is recommended.

In summary, PRISm is a complex state of pulmonary function limitation, the course and prognosis of which vary from person to person. Despite the presence of an association with COPD and a correlation with SAD, effective treatments are currently lacking. Future research needs to focus on more effective treatment strategies for PRISm to improve long-term health outcomes for patients.

Conclusions

Patients with PRISm have already exhibited symptoms and/or functional and/or structural abnormalities, manifesting as a state of airflow limitation. Early diagnosis of individuals at risk for PRISm can result in improved management and treatment strategies. Screening for PRISm includes conducting early lung function tests and/or CT scans of the chest among those who are at risk. However, since lung function tests provide limited information, imaging becomes particularly crucial for accurate detection and assessment. Imaging features, especially quantitative CT, provide valuable information in the study of PRISm to help identify disease subtypes, assess disease progression, including the evaluation of some PRISm co-morbidities, to better understand their pathophysiologic mechanisms and potential therapeutic targets, and thus guide clinical management. They are also related to the long-term prognosis of patients which have the potential to be regarded as an independent marker in longitudinal studies of PRISm. Closer monitoring and possible interventions may be required for those patients characterized by higher air trapping and emphysema. It is conceivable that future recommendations for a PRISm definition would include recognition of symptoms and more CT, MRI and X-ray imaging features in addition to spirometric criteria. In summary, the diagnosis and staging of PRISm require further exploration. More detailed research is needed in the future regarding early diagnosis and prognostic factor analysis for PRISm, especially in the field of imaging with quantitative, functional assessment and AI.

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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