Chronic Obstructive Pulmonary Disease (COPD): “Not a Cigarette Only Pulmonary Disease”
Augustine KH Tee, MBBS, FCCP, FRCP (Edin & Glas)

Since 2002, World COPD Day is celebrated yearly in November to raise awareness of Chronic Obstructive Pulmonary Disease (COPD) globally. ‘The Many Faces of COPD’ is the World COPD Day 2017 theme, chosen by The Global Initiative for Chronic Obstructive Lung Disease (GOLD), a World Health Organization collaborative. This serves a simple and timely reminder of the multifaceted challenge faced by healthcare professionals, and more importantly by COPD sufferers and their caregivers. Even with the explosion of literature on COPD in the last two decades, much is still uncertain regarding its pathophysiology, diagnosis, optimal treatment and prognosis in a disease that is increasingly recognised as a heterogenous entity.

In 1971, the first description of COPD in Singapore had 54 Chinese opium smokers who were all cigarette smokers. Their morbid anatomy, however, differed from pure cigarette smoking bronchitis and varied from bronchiectasis, emphysema to peri-bronchiolar fibrosis. Physiological abnormalities were varied among the individuals.

This early publication hinted at the non-uniform nature of the disease. Indeed, the standard definition of COPD by GOLD has changed every few years since the first GOLD report in 2001. GOLD 2017 defines COPD as “a common, preventable and treatable disease that is characterised by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious gases or particles”. Past definitions included terms such as ‘chronic inflammation’ and ‘irreversible airway obstruction’ which are inconsistently present in COPD, while demarcation into its two traditional distinct types of blue bloaters with chronic bronchitis versus emphysematous pink puffers appear too simplistic.

Based on projections, COPD will be the third leading cause of death worldwide by 2030. Projected prevalence in Asia will be 3 times higher than the rest of the world. Despite this, accurate population prevalence rates have not been available to aid healthcare resource planning and guide government medical spending. Local data estimates prevalence to be between 3.5% to 5.9%. Underdiagnosis is common, due to lack of public awareness and low usage of spirometry, which is an essential component of COPD diagnosis. A public survey conducted locally in 2015 during World COPD Day revealed that 65% have never heard of COPD. Of those who have heard, 90% were unaware of what COPD is, 28% incorrectly identified the commonest symptoms, 25% incorrectly identified COPD as a contagious disease, and 12.5% had the misconception that current or ex-smokers are not at a higher risk of COPD. In the face of such figures, COPD continues to be among the top 10 commonest cause of death in Singapore, and is a significant burden to our public health system. In a study from two public health clusters here in 2011, mean total cost was approximately USD$9.9 million per year with inpatient admissions being the major/main cost driver, contributing an average of USD$7.2 million per year.

Is the prevention of COPD solely a matter of controlling cigarette smoking in the population? Evidence suggests that although cigarette smoking is the main risk factor for developing COPD, it is by far not the only risk. Tobacco in the form of cigar, water pipe and marijuana may be associated with similar risk. Chronic exposures to noxious vapour, gas, dust or fumes, especially in “dusty trades” are also associated. Cigarette smokers without COPD have higher prevalence of respiratory symptoms such as cough, and demonstrate mild lung function abnormalities. Conversely, less than 50% of smokers have COPD.

About 10% of COPD patients have never smoked cigarettes. These patients tend to have milder forms of COPD with lower symptom severity, but nevertheless have similar prognosis to smokers with COPD. Hereditary alpha-1 antitrypsin deficiency and indoor air pollution from biomass are less relevant in urban Singapore. The influence of gender, age, occupational exposure, low socioeconomic status, low birth weight and severe childhood respiratory infections are known to place an individual at risk of acquiring COPD in adult life. Previously, it was thought that COPD developed due to a risk factor triggered accelerated lung function decline, starting from a maximally acquired adult lung function. A 2015 study on three large independent cohorts had studied the Forced...
Expiratory Volume in 1st second (FEV₁) decline over about two decades. It shed light for the first time that lung function trajectories may follow different paths leading to COPD. Half of the patients followed a well described trajectory of accelerated decline in lung function, but about half of the patients started with a low FEV₁ in early adulthood followed by a normal rate of lung function decline. Genetic and prenatal factors can also play a role in giving rise to the early origins of airflow limitation, as do severe childhood asthma theoretically depicted in a 3-phase lung function decline, consisting of a lower than predicted lung function at birth, early childhood FEV₁ decline due to poor asthma control, followed by a more physiological decline in later adult life. These concepts imply that COPD as a disease may begin in early life. As we explore the possibilities of interventions in early life, let us not forget that smoking cessation continues to be an intervention that can positively impact survival of COPD patients.

The need to investigate evolving risk factors do not negate the importance of other aspects of COPD care. Managing exacerbations, evaluating for comorbidities, pharmacotherapeutic advances, inhaler device management, patient phenotyping and biomarker endotyping are all aligned towards a move to personalised and precision medicine in COPD. Innovations in big data management and real-world effectiveness studies are under way to better utilise the interconnectivity of information systems in our public institutions. It is indeed an exciting time for the disease that is not “cigarette only”.

REFERENCES

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