COPD MANAGEMENT IN PRIMARY CARE:
WHAT ARE THE ISSUES AND WHAT CAN WE DO TO PROVIDE OPTIMAL CARE?

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INTRODUCTION

Chronic obstructive pulmonary disease or COPD, a chronic inflammatory airway disease, is one of the top 10 causes of mortality and reason for hospitalisation in Singapore. Apart from asthma, COPD is the second most common chronic respiratory disease being managed in primary care and is set to increase in prevalence due to the aging population in the local community. The Ministry of Health of Singapore has published clinical practice guidelines in 2006 to support respiratory and family physicians in providing optimal care for this group of patients based on clinical evidences. With growing amount of data and literature from research carried out globally, we examined the issues and use of evidence-based practice in managing three COPD patients with varying needs, in a local primary care centre.

CASE 1

Madam S is a 72-year-old Malay lady who is a non smoker and non drinker. She lives with her husband and son and is independent in her activities of daily living. She has a history of asthma since young and was diagnosed with bronchiectasis in 2004 at a tertiary hospital. A spirometry done in 2004 showed moderate airway obstruction; Forced Expiratory Volume in 1 second (FEV1) of 52%, Forced Vital Capacity (FVC) of 88% and the ratio of FEV1/FVC of 62% (refer to figure I).

In addition, Mdm S was also diagnosed with gastro-oesophageal reflux disease (GERD) in 2004. In 2005, she had pulmonary tuberculosis and completed anti-TB treatment in 2006. Between 2006 and 2007, she was admitted on 3 occasions for acute infective exacerbations of COPD. She was discharged well and subsequently followed up at a polyclinic.

Madam S was managed by the respiratory physician and she is on the following medications:

i) Budesonide/Formoterol [Symbicort] 320/9 mcg (2 puffs) bd
ii) Ipratropium [Atrovent] 40 mcg (2 puffs) tds
iii) Theophylline SR 125 mg bd
iv) Salbutamol 200 mcg (2 puffs) qds/prn

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Madam S had previously tried Tiotropium [Spiriva] 18mcg every morning for approximately 6 months but she had reported no improvement in symptoms although she may not have had been totally compliant with Tiotropium. Tiotropium was subsequently discontinued. She is also taking Omeprazole 20 mg bd and Domperidone 10mg tds for GERD.

CASE 2

Mr C is a 73-year-old Chinese man with diabetes mellitus, hypertension, hyperlipidemia and eczema. He was first diagnosed to have COPD in 2002 and the latest spirometry in March 2009 indicated severe disease (FEV1 42% of predicted). Based on the Global Initiative for Chronic Obstructive Lung Disease (GOLD) classification, FEV1 of less than 50% predicted is classified as severe stage III COPD[8].

He is on the current medications:

i) Salmeterol/Fluticasone [Seretide] 25/125 mcg (1 puff) bd
ii) Ipratropium [Atrovent] 40 mcg (2 puffs) tds
iii) Theophylline SR 125 mg bd
iv) Salbutamol 200 mcg (2 puffs) qds/prn

He continued to have multiple episodes of cough and shortness of breath with an average of 1-2 COPD exacerbations in a year. Mr C is a chronic smoker, and continued to smoke about 10 cigarette sticks a day, despite repeated advice to quit smoking by healthcare workers, including doctors and nurses in the polyclinic. During this time he was noted to be ambivalent in smoking cessation and swinged between the pre-contemplation and contemplation phases as assessed by the healthcare workers.

Mr C gradually reduced his cigarette smoking from 10 to 5 sticks a day. He agreed to a quit date set by the healthcare team. Initially Mr C found it hard to quit smoking as he would be beset with strong cravings and a sense of anxiety and fear. The family physician prescribed him with Varenicline (@Champix by Pfizer) for 2 weeks to reduce these withdrawal symptoms and to support the smoking cessation efforts but he was not compliant with the medication.

Mr C continued to smoke 4 to 5 sticks daily after the quit date. The polyclinic physician continued to assist Mr C with tips to continue reduction of cigarettes and advice on ways to minimise withdrawal side-effects. He finally succeeded in quitting smoking six months after the initial quit date.

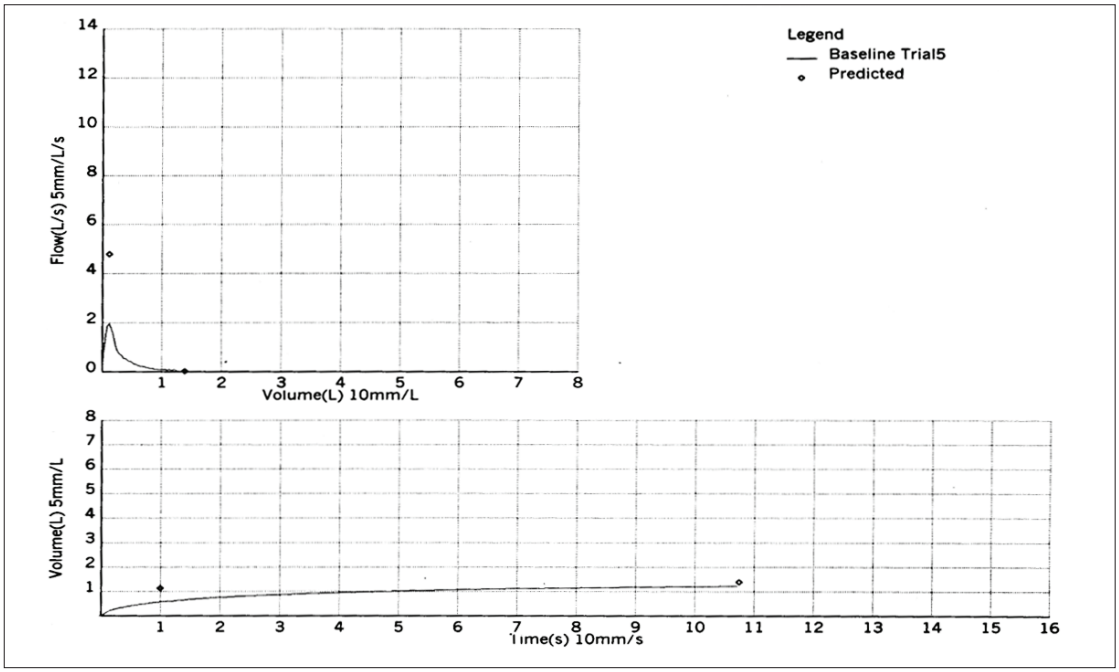
Fig I: Spirometry of Madam S

Test Results **Your FEV1 is 52% Predicted**

Parameter	Baseline Trial5	Trial3	Trial1	Pred	%Pred
FVC(L)	1.23	1.17	0.70*	1.39	88
FEV1(L)	0.58*	0.56*	0.57*	1.12	52
FEV1/FVC	0.47*	0.48*	0.81	0.76	62
PEF(L/s)	1.93*	1.63*	1.81*	4.79	40
MEF25-75(L/s)	0.19*	0.20*	0.48*	2.39	8
MEF75(L/s)	0.67*	0.68*	1.07*	4.54	15
MEF50(L/s)	0.20*	0.20*	0.56*	2.99	7
MEF25(L/s)	0.08*	0.11*	0.28*	0.91	9
FET(s)	10.75	9.78	2.74	-.--	--

*** Indicates Below LLN or Significant Post Change**

Baseline FEV1 Var=0.01L 2.6%; FVC Var=0.06L 4.6%; Session Quality A
Interpretation Moderate Obstruction



CASE 3

Mr T is a 75-year-old Chinese gentleman who is a non-smoker and non-drinker. He has a known history of previous pulmonary tuberculosis with bronchiectasis and fibrothorax diagnosed in 2002 based on a CT scan of his thorax. A recent chest X-ray (refer to figure II) showed clearly these two conditions clearly. He was first diagnosed with COPD GOLD stage 2 in 2002 when he was first admitted to a regional hospital for breathlessness. He was subsequently started on home nebulised bronchodilator therapy since 2002. Spirometry in 2002 showed a mixed restrictive and obstructive pattern.

In 2003, a lung function test was repeated in the regional hospital in view of his persistent symptoms and repeated admissions. It showed moderate airflow limitation with no significant bronchodilator response. Mr T then defaulted follow-up with the respiratory clinic in the hospital due to financial constraints and has been seeing the polyclinic for his follow-up. He had multiple hospitalisations for recurrent COPD exacerbations and consulted the polyclinic for the same reason on many occasions.

In November 2007, Mr T was started on long-term oxygen therapy (LTOT) during one of his hospitalisations. He receives 16 hours of oxygen delivered by nasal prongs at home daily. In

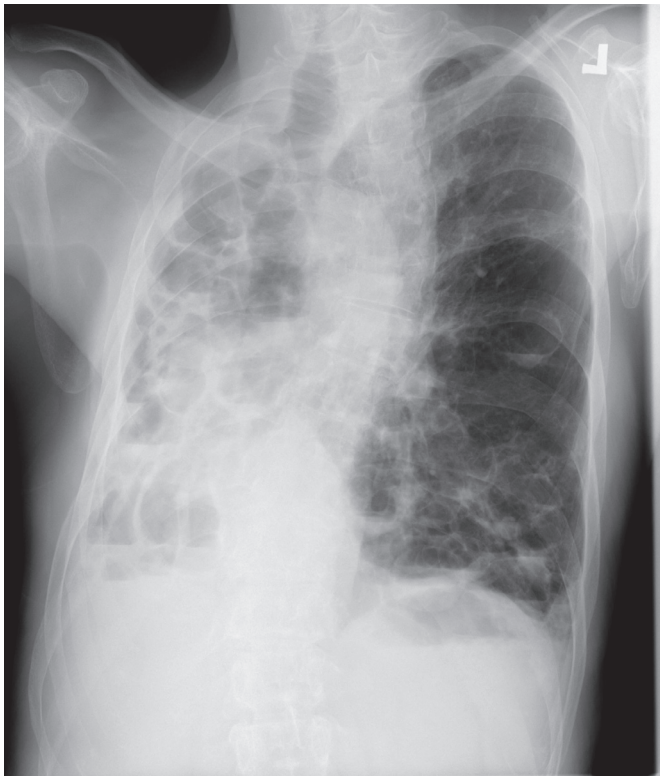


Fig II: Chest X-ray for Mr T

view of his financial constraints, he was referred for a medical social worker review. Subsequently, Mr T had waiver of charges for his medications and consultations at the polyclinic.

Mr T is noted to have declining weight over the past 6 months (refer to figure III). Examination showed a barrel chest with coarse crepitations and rhonchi over his lungs on auscultation on most occasions of his consultations. Pulse oximetry showed his blood oxygen saturation hovering between 92 to 93% when Mr T was at rest.

Fig III: BMI records for Mr T

Date	Weight (kg)	BMI
02/01/2009	49.5	20.4
30/01/2009	48.9	20.1
09/04/2009	47.9	19.7
22/05/2009	48.1	19.8

Mr T is currently on the following medications:
i) Budesonide [Pulmicort] 800 mcg (4 puff) bd
ii) Ipratropium [Atrovent] 40 mcg (2 puffs) tds
iii) Theophylline SR 250 mg bd
iv) Salbutamol 200 mcg (2 puffs) qds/prn

Despite compliance with medications, Mr T's COPD control has been suboptimal with 13 rescue therapies with aerosolized bronchodilator at the polyclinic over the past year and 1 hospitalisation over the same period.

DISCUSSION

CASE 1 - Issues encountered in managing Madam S

1. Can COPD develop in a non-smoking asthmatic patient?

A diagnosis of COPD should be considered in any patient more than 35 years old, who has chronic cough, sputum production, or dyspnoea and/or a history of exposure to risk factors for the disease. Spirometry is useful for the definitive diagnosis of COPD and staging of disease. It should be performed in individuals with risk factors and/or symptoms suggestive of COPD. Bronchodilator reversibility testing can help to distinguish asthma from COPD.1 Among non smokers, physician-diagnosed asthma increased the risk of mild and especially of moderate to severe COPD. Independently of asthma, risk of mild COPD in non smokers increased with age. In patients below age 60, the risk is lower in men than in women[2].

The primary risk factor for development and progression of COPD is smoking. However less than 25% of smokers develop COPD and about 15% of COPD related mortality occurs in non smokers. Other factors which have been identified to be important in the development and progression of COPD include occupation, genetic factors (alpha 1 antitrypsin), air pollution, sex, socioeconomic status, nutrition and childhood exposures[3].

2. Is Budesonide/Formoterol [Symbicort by Astra Zeneca] effective in COPD treatment?

Recent studies have shown that Budesonide/Formoterol turbuhaler resulted in significantly improvement in pulmonary function. Two large pivotal trials, the 6 month SHINE and 1 year SUN trials have shown that inhaled combination therapy is superior to either agent alone in the treatment of chronic obstructive pulmonary disease[4]. In these studies, Budesonide/Formoterol reduced the mean number of severe exacerbations per patient per year by 24% versus placebo and 23% versus Formoterol. Increase in FEV1 of 15% versus placebo and 9% versus Budesonide was noted. The morning Peak Expiratory Flow (PEF) improved significantly on day 1 versus placebo and Budesonide; after 1 week, morning PEF was improved versus placebo and Budesonide/Formoterol. These improvements in the morning and evening PEF versus comparators were maintained over 12 months[5]. Combination formulation in a single inhaler will be more convenient and will improve compliance in patients. Recent clinical trial shows that Budesonide/Formoterol added to Tiotropium improves lung function, health status, symptoms and morning activities in COPD patients[6].

3. Why does a clinically proven drug deem to be "ineffective" in patient's treatment?

Madam S was initiated on Tiotropium [Spiriva by Boehringer Ingelheim] but reported little improvement in her symptoms. However, Tiotropium has been recognised to benefit COPD patients in many ways. A recent trial, UPLIFT which included 5993 patients who were followed for 4 years compared the effect of usage of Tiotropium with control. The study shows that Tiotropium significantly delayed time-to-first exacerbation (16.7 versus 12.5 months) and time-to-first hospitalisation for exacerbations and reduced the mean number of exacerbations by 14%[7].

Family physicians need to probe deeper into the apparent "failure" of a drug with evidence based effectiveness. The factors can be divided into the following domains: patient, doctor/physician, disease/treatment and healthcare system[8].

1. Patient
   a. perception and expectation of effect of medication
   b. adherence to prescribed therapy
2. Doctor/physician
   a. doctor-patient rapport and mutual trust
   b. effective communication skills to relate disease etiology and effect of drugs
3. Disease/treatment
4. Healthcare system

We need to understand Mdm S's perception and expectation of Tiotropium, when it was started as part of her treatment regimen. The drug would likely reduce her frequency of exacerbation and hospitalization but would not abolish all her symptoms. In general, lung function would decline for any

COPD patient with time, irrespective of any concomitant trigger, due to aging. It is part of progression of the disease. Nonetheless, reducing the rate of deterioration of her lung function with Tiotropium, would not be apparent to Mdm S. It is important to educate the patient on the disease pattern and specific effect of the drug at its commencement, so that the expectation is set at the appropriate level. Details should be communicated and doubts are clarified at the same setting. This will be facilitated if doctor-patient relationship has been well established.

Mdm S's adherence to treatment may also have been hindered by the high cost of Tiotropium. Nonetheless, a local study conducted in 2006 had shown significant cost savings in favour of Tiotropium[9]. These apply to the incremental cost per year of Tiotropium treatment, to the cost savings in hospitalisation per year and to the cost required to reduce one hospitalisation. There is a potential overall cost saving of 83% after the addition of Tiotropium to the medical regimen of patients with COPD[9]. This information should be communicated to the patient so that she can make appropriate informed decision.

There are avenues to support Mdm S's treatment if cost remains an issue. Medical social worker in the polyclinics can evaluate her financial status and for the socioeconomically disadvantaged patients, treatment cost can be waived or further subisdised. COPD patients can also tap into their or their family member's Medisave funds to support their treatment[10]. The Medisave scheme is available in polyclinics and approved GP clinics in the community.

CASE 2 - Issues encountered in managing Mr C

1. Does COPD patient understand the link between smoking and their diseased lungs and how does it impact on the patient's outcome?

It is the duty of the doctor to assess and advise the patient to quit smoking. Smoking is a major risk factor for COPD, and it has been estimated that about 10 to 30% of smokers will develop COPD[12,13]. It has also been shown that smoking cessation is the main preventive measure that reduces the decline in lung function for all patients at various stages of COPD[14,15].

This should be communicated to the patient and translate this technical information into the patient's context, in the form of personal benefits that are easily understood by a layperson such as Mr C. In this case, Mr C has been counselled by both the family physician and his team of nurse counsellors. The healthcare team provided him with a reflection of his current health status with recurrent episodes of dyspnoea, and contrasted it with a future state of improved lung function (after smoking cessation), when he can participate in more physical activities. Other benefits in the "future state" in terms of cost savings from cessation of cigarettes purchase and reduced healthcare expenditure due to improved health status, further enhanced this strategy to promote smoking cessation.

Concurrently, Mr C's stage of readiness using the behaviour change model8 to quit smoking, was classified and appropriate action was taken at each consultation. This is one way of gauging the progress and consistently supporting this patient towards smoking cessation.

2. What clinic processes can be introduced or implemented to facilitate the family physician in managing the patient's smoking habit?

The United States Clinical Practice Guidelines on Treating Tobacco Use and Dependence (2008)[16] recommend the implementation of an office-wide system that ensures that for every patient, at every clinic visit, tobacco-use status is queried and identified. This could take the form of a change in office system, such as the inclusion of tobacco-use status into templates on patient's vital signs.

System re-design has been implemented in a local cluster of polyclinics to facilitate the capture of smoking related information for at risk patients, including those with COPD. For Mr C, his smoking status was determined by trained health care attendants at the health monitoring station in these polyclinics at every visit and recorded into the IT system. This information is transmitted to the attending doctor in the consultation room via the system, which prompts and reminds the doctor to address the smoking habit.

In America, such behavioural counselling interventions reach about 70 percent of smokers in primary care, who visit their doctors each year and result in 5 percent to 10 percent overall quit rates[17]. The full impact of these interventions can best be seen when they are employed widely.

CASE 3 - Issues encountered in managing Mr T

1. In what ways can a family physician assess and classify the COPD status of a patient and to decide when the patient requires a review by the pulmonologist?

From history, the family physician can identify patients at high risk of COPD. These include patients who are above 35 years old, who are smokers or were ex-smokers. These patients would have exertional shortness of breath, chronic cough, regular sputum production, wheeze and frequent 'winter bronchitis'. There must also be no suggestion of asthma[18].

However, this is not adequate and the family physician still requires a spirometry to provide an objective measure of COPD. Spirometry is useful for the definitive diagnosis of COPD and for staging of COPD[1]. The NICE COPD guidelines recognised that not all family physicians have spirometry equipment, thus a suggestion was made for better and direct access to lung function laboratories by family doctors[18].

Specialist opinion was still required to confirm diagnosis and to optimise therapy in patients with severe COPD, or those with complication of cor pulmonale. Specialist opinion on the need for oxygen therapy may be required in patients with severe COPD. Another category of patients belonged to those with uncertain diagnosis, where help with diagnosis is crucial to ensure optimal outcome[18].

2. *How do we advise patients to improve on their nutrition status?*

Nutritional status in COPD patients is important because of greater resting energy expenditure required to maintain an adequate breathing effort. Further impediments to eating such as shortness of breath, chronic sputum production, chronic cough, depression and medication side effects also have an impact on oral caloric intake. Hence due to overall increased energy requirements, patients can loss weight. This is associated with impaired pulmonary status, reduced diaphragmatic mass, lower exercise capacity and higher mortality rates[19].

Patients should be advised to take smaller but frequent meals with softer and easy-to-chew foods to reduce the effort of eating. Foods that can 'give gas' such as carbonated drinks, fried, greasy or heavily spiced foods, beans, cabbage, cauliflower, should be avoided to reduce fullness. It may be necessary for patients to use their bronchodilator prior to meals. Adequate fluids such be taken to thin mucous secretions. Meals should be simple to prepare to reduce exertion during preparation. Salt intake should also be limited. Use of nasal prongs is recommended should continuous oxygen is prescribed[20, 21, 22]. (refer to figure IV)

Figure IV: Tips On How COPD Patients Can Improve Their Nutritional Status

i) Choose from 4 groups of food:

Milk and milk products 2 servings

Fruit and vegetables 5 servings

Meat and alternatives 2-3 servings

Bread and cereals 5 servings

ii) To help manage shortness of breath when eating:
- Eat smaller, frequent meals (5-6 meals or snacks/day). Too much food at one time is overwhelming. Frequent, smaller meals help improve poor appetite.
- Try softer, easy-to-chew foods to reduce effort of eating.
- Rest before meals. Eat slowly in a relaxed pace.
- Sip fluids, do not gulp air and avoid foods that can "give gas". Examples include:
  - Carbonated drinks
  - Fried, greasy or heavily spiced foods
  - Apples, avocados and melons
  - Beans, broccoli, brussels sprouts, cabbage, cauliflower, corn, cucumbers, leeks, lentils, onions, peas, peppers, pimentos, radishes, scallions, shallots and soybeans
- Excess gas can cause fullness.
- Take bronchodilator inhalers before meals. This helps improve breathing during meals.

iii) Fluids:
- Try to drink 6-8 cups of fluids such as water, fruit juices or milk each day. Adequate fluids help thin mucous secretions.
- Milk and diary products do not produce more mucous but can coat mucous already present and make it more noticeable. Try drinking citrus juice after taking dairy products to help the mucous.
- Limit fluid taken with meals. Have most liquids after or between meals.

iv) Tips to save time and energy:
- Make "easy to prepare" one dish meals; make extra and freeze some for later.
- Use the oven, microwave, or toaster oven if stove-top cooking is tiring.
- Try ready-to-serve meals available in the deli or frozen sections.
- Try services such as Meals on Wheels.
- Plan and prepare ahead of time as much as possible.

v) Limit salt intake:
- Use herbs or no-salt spices to flavour food.
- Don't add salt to foods when cooking.
- Read food labels and avoid foods with more than 300 mg sodium per serving.

vi) Use nasal prongs while eating if continuous oxygen is prescribed. Eating and digestion requires oxygen so the body will need the extra oxygen.

vii) If patient is underweight:
- Being underweight increases risk for illness.
- Eat small meal 5-6 times per day.
- Include protein foods at all meals, i.e. milk, cheese, yogurt, eggs, meat, poultry, fish, nut, butters, legumes, soy products.
- Drink liquids with calories, i.e. milk, milkshakes, juices, Ensure. Drink them after meals.
- Eat the main meal first.
- Weekly weighing to ensure no further weight loss.

viii) If patient is overweight:
- Being overweight makes breathing more difficult. To lose weight, change eating and exercise habits gradually.
- Reduce portion sizes, continue to have 2-3 servings of protein per day.
- Limit high fat and high sugar foods, eat more fruits and vegetables instead.
- Increase activity as tolerable.
- Don't keep 'treats' at home that can encourage snacking.
- Weekly weighing to track weight loss.

CONCLUSIONS

These 3 cases illustrate the breadth of issues encountered in managing patients with COPD.

- Case 1 illustrates the issues of diagnosis of COPD to be considered in any patient more than 35 years old who has a chronic cough.
- Case 2 illustrates the issues encountered in managing a patient who continues to smoke.
- Case 3 illustrates the issues in a patient with poor COPD control and in what way we can assess, classify and advise the patient to improve his health status, deal with acute exacerbations and consider a written action plan.

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