Smoking Cessation Programs and Other Preventive Strategies for Chronic Obstructive Pulmonary Disease

Raj Kumar*, VK Vijayan**

Introduction

Chronic obstructive pulmonary disease (COPD) is a major public health problem worldwide. It is the fourth leading cause of chronic morbidity and mortality in the United States and is projected to rank fifth in 2020 as a worldwide burden of disease according to a study published by the World Bank/World Health Organization. Tobacco smoking is the main aetiological factor of COPD. Apart from COPD, smoking is a risk factor for other lung diseases such as pneumonia, tuberculosis, asthma, interstitial lung diseases, pneumothorax and lung cancer etc. The International Classification of Disease (ICD-10) has recognized that “Tobacco Dependence” is a disease. The medical profession especially in underdeveloped countries has not taken a serious view of the ‘disease of tobacco dependence’, and has not made serious attempts to treat the disease, ‘Tobacco Dependence’. In many countries, air pollution resulting from the burning of wood and other biomass fuels has been identified as a COPD risk factor. Occupational exposures to dusts, gases, fumes and irritants are also important risk factors for COPD. Smoking cessation is the most important strategy for the prevention and progression of COPD. A number of reviews and guidelines on smoking cessation have recently been published. They provide recommendations for interventions and strategies to promote the treatment of tobacco dependence. The current situation calls for integrating smoking cessation as a routine strategy in chest clinics. This puts an onerous responsibility on the physicians to have detailed knowledge about smoking cessation methods, and the use of medication. Others methods for COPD prevention include prevention of environmental tobacco smoke exposure and reduction of indoor and outdoor air pollutants.

Smoking Prevention

Public health programs and policies that focus on tobacco-use prevention are critically important. It should be delivered to people through various channels such as health care providers, community activities, and mass media such as radio, television and news papers. Health education regarding adverse effects of tobacco use should be addressed to the students in schools and colleges. Smoking cessation activities should target all ages including young children, adolescents, young adults, and pregnant women. Interventions to prevent smoking uptake and maximize cessation should be implemented at every level of health care system. Making all public places smoke-free should be encouraged.

Smoking Cessation

Smoking cessation is the most effective strategy for prevention of COPD. Quitting smoking can prevent or delay the development of airflow limitation, or reduce its progression, and can have substantial effect on subsequent mortality. Counselling alone or counselling with medication is used for tobacco cessation. Significantly high continuous abstinence rates are seen with the medications as compared to the counselling alone.

Counselling

Counselling delivered by physicians and other professionals significantly increases quit rates over self-initiated strategies. Even a brief (3-minute) period of counselling to urge smoker to quit results in smoking cessation rates of 5-10%.

National cancer Institute, USA has formulated brief strategies to help the patients willing to quit and this includes a “5A” (ask, advise, assess, assist and arrange) based intervention in a primary care set up.

i. “ASK” the patients about tobacco use and identified at each visit. Tobacco use status is queried and documented, and general and vital informations are obtained.

ii. “ADVISE” to the patient should be clear, strong and personalized according to the patient’s current health/illness, motivation level or impact on children in the household.

iii. “ASSESS” the willingness of the patient to quit. Provide motivational assistance to those unwilling to quit and provide additional information in special situations such as adolescence and pregnancy.

iv. “ASSIST” the patient with a quit plan. Set a quit date within the next two weeks. Patient is advised to tell friends, family and co-workers about quitting and request understanding and support. Anticipate challenges to quit attempt and educate about nicotine withdrawal symptoms. Remove all tobacco products from environment and avoid places associated with smoking. Provide practical counselling (problem solving and skill training). Total abstinence is essential. If the patient had past quit experiences, identify what helped and what hurt. Anticipate triggers or challenges in upcoming attempts. Alcohol use to be minimized and tell the patient about risk of relapse if he/she continues to drink. Encourage housemates to quit smoking or not smoke in subject’s presence. Provide additional information in special situations such as adolescence and pregnancy.

Pharmacotherapy

Pharmacotherapy is indicated when counselling is not sufficient to help patients quit smoking. Pharmacotherapies
can be divided into replacement therapy, antagonist therapy, therapies to make drug intake aversive and non-nicotine medications that mimic nicotine effects. Various drugs used are shown in Table 1.

Nicotine Replacement Therapies (NRTs) are the appropriate first line agents. Numerous studies indicate that nicotine replacement therapy in any form (nicotine gum, inhaler, nasal spray, transdermal patch, sublingual tablet, or lozenge) reliably increases long-term smoking abstinence rates.12,22,23 nicotine replacement therapy is more effective when combined with counselling and behaviour therapy.24 For those smoking 1-24 cigarettes per day, 2mg gum and for those smoking >25 cigarettes per day a 4mg gum is recommended. One gum to be chewed for 30 minutes every 1 or 2 hours and not more than 20 lozenges per day.

**Table 1 : Various drugs used in tobacco dependence**

<table>
<thead>
<tr>
<th>Pharmacotherapy</th>
<th>Side Effects</th>
<th>Dosage and Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bupropion SR</td>
<td>Insomnia</td>
<td>150 mg every morning for 3 days, then 150 mg twice daily, 7-12 weeks and up to 6 months</td>
</tr>
<tr>
<td></td>
<td>Dry mouth</td>
<td></td>
</tr>
<tr>
<td>Nicotine Gum</td>
<td>Mouth soreness</td>
<td>1-24 cigs/day-2mg gum [to up to 24 pcs/day]</td>
</tr>
<tr>
<td></td>
<td>Dyspepsia</td>
<td>25 cigs/day-4mg gum [Up to 24 pcs/day] Up to 12 weeks</td>
</tr>
<tr>
<td>Nicotine Lozenge</td>
<td>Nausea</td>
<td>If patient smokes more than 30 min. after waking -2 mg lozenge</td>
</tr>
<tr>
<td></td>
<td>Insomnia</td>
<td>If smokes less than 30 min. after waking</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 mg lozenge</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Not more than 20 lozenges per day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Up to 12 weeks</td>
</tr>
<tr>
<td>Nicotine Inhaler</td>
<td>Local irritation of mouth and throat</td>
<td>6-16 cartridges/day Each cartridge is 4mg in 80 inhalations Up to 12 weeks</td>
</tr>
<tr>
<td>Nicotine Nasal Spray</td>
<td>Nasal irritation</td>
<td>-0.5 mg in each nostril 1 to 2 doses per hour 8-40 doses/day for a period of 3 to 6 months</td>
</tr>
<tr>
<td>Nicotine Patch</td>
<td>Local skin reaction</td>
<td>21 mg/24 hours for 4 weeks</td>
</tr>
<tr>
<td></td>
<td>Insomnia</td>
<td>14 mg/24 hours for 2 weeks</td>
</tr>
<tr>
<td>Varenicline</td>
<td>Mild to moderate nausea</td>
<td>0.5 mg once a day for 1-3 days 0.5 mg twice a day for 4-7 days 1 mg twice a day from day 8 to end of treatment which is for a duration of 12 weeks</td>
</tr>
</tbody>
</table>

A. Medications that mimic nicotine effects can be used as second line pharmacotherapy.

i. **Bupropion SR**: Bupropion SR is a non-nicotine medication with abstinence rate twice that of the placebo.25 It is very effective and cheap treatment for nicotine addiction in smokers.26 It acts by blocking the neural uptake of dopamine and noradrenaline. The drug is generally well tolerated in cardiovascular disease patients and can be used in pregnancy if a favourable risk benefit ratios exists. Insomnia (35-40%) and dry mouth (10%) are most common adverse events. It is contraindicated in seizure disorders, eating disorders and if patient has used MAO inhibitor in past 14 days. It is given in a dose of 150mg OD for 3 days then 150 mg BD is continued for next 7-12 weeks following the quit date which is preferably between the first or second week of treatment. Consider giving maintenance therapy for 6-12 months in selected patients. It has been found to be associated with minimal post cessation weight gain and is to be preferred in patients with depressive ideation. It has proven to be superior to NRTS but has no added advantage if combined with them.

ii. **Varenicline**: Varenicline is a new nicotinic acetylcholine receptor partial agonist that aids smoking cessation by relieving nicotine withdrawal symptoms and reducing the rewarding properties of nicotine and it has been demonstrated to be safe and efficacious.27-29 The dose is 0.5 mg once a day for 1-3 days, then 0.5 mg twice a day for 4-7 days, followed by 1 mg twice a day from day 8 to end of treatment which is for a duration of 12 weeks.

iii. **Clonidine**: Clonidine is a postsynaptic 2 agonist that dampens sympathetic activity originating at the locus ceruleus. Clonidine in doses 0.1-0.4 mg/day for 2-6 weeks has been found to be useful. The most common side effects of Clonidine are dry mouth, sedation and constipation. Clonidine appears to increase the quit rates similar to nicotine replacement therapy.

iv. **Anxiolytics**: Anxiety is a prominent symptom of nicotine withdrawal. Smoking decreases some measures of anxiety and may reduce stress induced anxiety. So temporarily replacing the anxiolytic effects of nicotine with another medication during the first week of cessation might make the cessation easier. Diazepam and beta blockers have been widely used with mixed results. Buspirone is a serotoninergic agonist, which acts as an anxiolytic but produces mixed results, when used for smoking cessation. Advantage over the benzodiazepines being non-sedating and does not have an addictive potential. It is used in a dose of 15-30 mg/day in divided doses.
v. Antidepressants: Many antidepressants have been tried with varied results. These are helpful only when the patients have underlying depression.

vi. Stimulants: The aim is to replace the stimulant effects of nicotine (e.g., improved energy and concentration) with medication in the first week of cessation. The drugs that have been used are amphetamine and methylphenidate.

vii. Anorectics: Anorectics were initially to combat post cessation hunger and weight gain because these are the two most widely cited reasons for difficulty in stopping smoking. Encouraging results were obtained with fenfluramine and phenylpropanolamine in short term trials.

viii. Sensory Replacement: Black pepper extracts, capsaicin, denicotinised tobacco, flavourings all decrease cigarette craving and withdrawals. A citric acid inhaler has also been developed and showed some promise in two clinical trials.

ix. Acupuncture: Acupuncture technique is also used in smoking cessation. One common rationale for using acupuncture for smoking cessation is that acupuncture can release endorphins that assist in cessation.

The above mentioned methods have been used alone or in various combinations with mixed results. Ultimately it is the willingness/motivation of the patient which is of utmost importance in the success of smoking cessation. Only when the patient is willing to stop smoking, the physician can help him.

B. Antagonists - The goal of the antagonists is to prevent cigarettes from producing positive reinforcing and subjective effects.

i. Mecamylamine: Mecamylamine is a non-competitive blocker of both central and peripheral nervous system nicotine receptors that decreases the positive subjective effects of cigarettes. It does not precipitate withdrawal in humans, perhaps because it is a direct blocker.

ii. Naltrexone: Naltrexone is a long acting form of opioid antagonist naloxone. The rationale for using Naltrexone for smoking cessation is that the performance enhancing and other positive effects of nicotine may be opioid mediated. Couple of studies point out the beneficial effects of Naltrexone. Still a study with large sample size is lacking before actually using it in clinical practice.

C. Medication that make intake aversive: Medication in this class produces unpleasant events when the patient ingests the medication. Silver Acetate combines with the sulphides in tobacco smoke to produce a bad taste. Silver Acetate has been tested as a gum and as a pill. Compliance is very poor with this drug.

Air Pollution and Occupational Exposure Prevention

Air pollution, both indoor and outdoor, and occupational exposure to dust, smoke, and fumes are important risk factors for COPD. Efforts to reduce smoking through public health initiatives should also focus on passive smoking to minimize risk for non-smokers. Implementing, monitoring and strict enforcing of rules regarding control of airborne exposure in the workplace is an important step to prevent occupational exposure. Intensive and continuing education of exposed workers, industrial managers, health care workers, primary care physicians, and legislators should be initiated. Employers, workers, and policymakers should be educated on how smoking aggravates occupational lung diseases and why efforts to reduce smoking where a hazard exists are important.

To summarize, Most of the COPD cases are attributed to cigarette smoking. Occupation-related exposures, genetic factors, indoor and outdoor exposures to air pollutants also play a role. Thus, COPD largely can be prevented. Prevention of COPD begins with reducing and/or eliminating smoking initiation among teenagers and young adults and encouraging tobacco cessation among current smokers. Many people worldwide have been exposed to gases, vapours, fumes, and dusts that may cause COPD. Public health programs and policies that focus on tobacco-use prevention and cessation, reducing occupational exposure to dusts and chemicals, and reducing other indoor and outdoor air pollutants are critically important. In our country, biomass fuel used for cooking purposes is an important risk factor for the development of COPD. In order to prevent the development of COPD in this subset of individuals especially in females, efforts should be made to educate the people about the hazards of biomass fuel exposure and also to encourage them to use pollution free methods for cooking.

At the end the message is: “Even a single puff is detrimental to the quit attempt.”

References


11. National Institute for Clinical Excellence (NICE). Guidance on the use of nicotine replacementtherapy (NRT) and Bupropion for smoking


