Asthma and Cardiovascular problems: An overview
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Introduction
The management of cardiovascular diseases in a patient with asthma or chronic obstructive pulmonary disease is a common problem. It is made difficult by the direct asthma-inducing effect of some cardiovascular drugs and by potential toxicity resulting from drug-drug interactions. As an example, beta blockers should be used with great caution or not at all in patients with chronic asthma (including chronic obstructive pulmonary disease) or acute allergic or exercise-induced bronchospasm. The angiotensin converting enzyme inhibitors and sympathetic blockers also may have deleterious effects in this setting.

There are a number of modalities that may be beneficial in the management of the patient with acute myocardial infarction (MI). These include aspirin, direct angioplasty or thrombolysis, angiotensin converting enzyme (ACE) inhibitors, beta blockers, and, in most patients, statins. The administration of a beta blocker following an acute MI has been shown to reduce morbidity and mortality in multiple trials. If the patient tolerates the beta blockade, oral beta blocking drugs can then be used in patients of acute myocardial infarction with asthma.

Supraventricular arrhythmias, including AF, are common in patients with asthma and have adverse prognostic implications in patients with acute exacerbations of asthma. Rate control can usually be achieved safely with calcium channel antagonists in this setting. In patients with obstructive pulmonary disease who develop AF, a calcium channel antagonist agent (diltiazem or verapamil) is preferred for ventricular rate control.

ACE inhibitors may be inappropriately stopped in patients with pulmonary causes of cough in patient with heart failure. If the patient tolerates the beta blockade, can then be used in patients of heart failure with asthma.

Indexing words
Asthma, Hypertension, Ischemic Heart Disease, Arrhythmia, Heart failure

Hypertension
The management of hypertension in a patient with asthma or chronic obstructive pulmonary disease (COPD) is a common problem owing to the high prevalence of each condition in the adult population. Beta-blockers should be used with great caution or not at all in patients with chronic asthma (including chronic obstructive pulmonary disease) or acute allergic or exercise-induced bronchospasm. Beta-blocker being made asthma worse, sometimes acutely. JNC 7 report mentioned beta-blocker should generally be avoided in individuals who have asthma. At present, ACE inhibitors are currently not first-line therapy in patients with asthma or COPD. It is safest to administer only low thiazide doses (12.5 to 25 mg of hydrochlorothiazide) to nonedematous hypertensive patients with asthma or COPD. The calcium channel blockers are excellent agents for the treatment of hypertension in asthma. Thus, the use of a calcium channel blocker alone or with a low dose of a thiazide diuretic represents the preferred regimen for the initial management of the hypertensive asthmatic. Steroid use in asthma commonly promotes fluid retention and hypertension, judgment is therefore necessary in deciding on an optimal dose.
Ischemic Heart Disease

Unless there are specific contraindications aspirin should be administered to all patients with acute coronary syndromes. Approximately 10% of asthmatic patients develop bronchoconstriction when given salicylates (e.g., aspirin). Aspirin can induce worsening of asthma, which can be severe, accompanied by urticaria. Aspirin sensitive asthmatics are usually adult with a history of nasal symptoms often due to nasal polyps. Clopidogrel should be administered in patients with acute coronary syndrome who are unable to tolerate aspirin due to hypersensitivity primarily manifested as asthma.

Beta-blocker should be avoided in subset of patients who have true bronchospastic lung disease, and use of nitrate and calcium antagonists is preferred. Since many of these patients receive medications for their pulmonary disease that may increase their heart rate or even produce supraventricular tachycardia, it is preferable to use a heart rate slowing calcium antagonist such as diltiazem or verapamil. Many patients with a history of only asthma or mild chronic obstructive pulmonary disease may be able to tolerate small doses of cardio selective beta blockers with careful monitoring. β2 agonist in high dose may worsen angina.

In unstable angina and non ST elevated myocardial infarction, patients with significant COPD who may have a component of reactive airway disease should be administered beta blocker very cautiously; initially, low doses of beta 1 selective agent should be used. If there are concerns about possible intolerance to beta blockers, initial selection should favor a short acting β1 specific drug such as metoprolol. Mild wheezing or a history of COPD mandates a short acting cardioselective agent at a reduced dose rather than the complete avoidance of a beta blocker.

Beta blockers are safe and effective in patients with mild pulmonary disease with MI. A survival benefit in patients with MI from beta blockade is seen with relative contraindications to such therapy such as chronic obstructive lung disease (COPD)/Asthma. Beta blocker therapy should probably be applied universally to all patients without absolute contraindications. History of asthma, severe chronic pulmonary disease are relative contraindication to beta blocker therapy, in a setting of MI the clinician has the option of assessing the effects of beta blockade with the short acting intravenous beta blocker esmolol. If the patient tolerates the beta blockade, long acting oral beta blocking drugs can then be used with increased confidence.

Arrhythmia

Supraventricular arrhythmias, including AF (Atrial Fibrillation) are common in patients with chronic obstructive lung disease and have adverse prognostic implications in patients with acute exacerbations of chronic obstructive pulmonary disease. Treatment of the underlying lung disease and correction of hypoxia and acid-base imbalance are of primary importance.

Theophylline and betaadrenergic agonists, which are commonly used to relieve
bronchospasm in these patients, can precipitate AF and make it difficult to control the rate of ventricular response. Beta-blockers, sotatol, propafenone, and adenosine are contraindicated in patients with bronchospasm and wheezing. Rate control can usually be achieved safely with calcium channel antagonists; digoxin offers no advantage over calcium channel antagonists in this situation. Pharmacological antiarrhythmic therapy and electrical cardioversion may be ineffective against AF until respiratory decompensation has been corrected. Intravenous flecainide may be efficacious in restoring sinus rhythm in some patients. Electrical cardioversion may be attempted in haemodynamically unstable patients. In resistant cases, AV nodal ablation and ventricular pacing may be necessary to control the ventricular rate. The role of anticoagulation in patients with AF due to chronic obstructive lung disease has not been studied specifically\textsuperscript{12}.

Recommendations for management of AF in patients with pulmonary diseases\textsuperscript{12}.

**Class I**
1. In patients who develop AF during an acute pulmonary illness or exacerbation of chronic pulmonary disease, correction of hypoxaemia and acidosis are the primary therapeutic measures. (Level of evidence: C)
2. In patients with obstructive pulmonary disease who develop AF, a calcium channel antagonist agent (diltiazem or verapamil) is preferred for ventricular rate control. (Level of evidence : C)
3. Attempt electrical cardioversion in patients with pulmonary disease who become haemodynamically unstable owing to AF. (Level of evidence : C)

**Class III**
1. Use of theophylline and beta-adrenergic agonist agents in patients with bronchospastic lung disease who develop AF. (Level of evidence: C)
2. Use of beta-blockers, sotatol, propafenone, and adenosine in patients with obstructive lung disease who develop AF. (Level of evidence: C)

**Heart failure**
Because dyspnoea is the key symptom in both HF and pulmonary disease, it is important to distinguish the two diseases and to quantify the relative contribution of cardiac and pulmonary components to the disability of the patient when both disorders co-exist. Some drugs used to treat HF can produce or exacerbate pulmonary symptoms. ACE inhibitors can cause a persistent nonproductive cough that can be confused with a respiratory infection, and conversely, ACE inhibitors may be inappropriately stopped in patients with pulmonary causes of cough. Therefore, physicians should seek a pulmonary cause in all patients with HF who complain of cough, whether or not they are taking an ACE inhibitor. The cough should be attributed to the ACE inhibitor only if respiratory disorders have been excluded and the cough disappears after cessation of ACE inhibitor therapy and recurs after reinstition of treatment. Similarly, beta-blockers can aggravate bronchospastic symptoms in patients with asthma, and therefore, all beta blockers (regardless of their selectivity) should be avoided in patients with reactive airways disease\textsuperscript{13}.

**Conclusion**
Patient of bronchial asthma with cardiovascular problem need special attention for management of both problems simultaneously. If tolerated beta blocker should be used in patient with IHD. Clopidogrel should be administered in patients with acute coronary syndrome who are unable to tolerate aspirin. In supraventricular arrhythmias calcium channel blockers are the drug of choice. Patient with heart failure and asthma beta blocker and ACE inhibitor also can be used if tolerated.
References