

# Antianginal Drugs

Angina medications are used for angina pectoris or chest pain. The types of chest pain are chronic stable angina (which is associated with atherosclerosis), unstable angina (early stage of progressive coronary artery disease), and vasospastic angina (which results from spasms in the layer of smooth muscle that surrounds atherosclerotic coronary arteries). The three main classes of drugs used to treat angina pectoris are nitrates and nitrites, beta-blockers, and calcium channel blockers.

The goal of the treatment is:

1. Minimize the frequency of attacks and decrease the duration and intensity of angina pain
2. Improve the patient's functional capacity with as few adverse effects as possible
3. Increase blood flow to ischemic heart muscle and decrease myocardial oxygen demand
4. Prevent or delay the worst possible outcome, which is a myocardial infarction

## **Nitrates and Nitrites**

Nitrates are available on many forms including sublingual, chewable tablets, oral capsules/tablets, IV solutions, transdermal patches, ointments and translingual sprays. They are broken down into rapid-acting forms and long-acting forms. The rapid-acting forms include sublingual and IV solutions. These are used to treat acute angina attacks. The long-acting forms are used to prevent angina episodes.

## **Mechanism of Action**

Nitrates dilate all blood vessels; however, they predominately affect venous vascular beds and have a dose-dependent arterial vasodilator effect. This vasodilation happens because of relaxation of smooth muscle cells.

1. Vasodilation results in reduced myocardial oxygen demand and therefore more oxygen to ischemic myocardial tissue and reduction of angina symptoms.
2. By causing venous dilation, the nitrates reduce venous return and in turn reduce the leftventricular end-diastolic volume (preload) and results in a lower left ventricular pressure.

### **Adverse Effects**

Headache is the most common undesirable effect. If nitrate-induced vasodilation occurs too rapidly, reflex tachycardia occurs which is characterized by an increase in heart rate. This is because the cardiovascular system tries to overcompensate. Other adverse effects include postural hypotension and tolerance may develop.

### **Beta-Blockers**

The beta-blockers that are classified as antianginal are atenolol, metoprolol (Lopressor), propranolol (Inderal) and nadolol (Corgard).

### **Mechanism of Action**

Beta blockers block beta1-receptors on the heart. This allows:

1. Decrease in heart rate which decreases myocardial oxygen demand and increases oxygen delivery to the heart.
2. Decreases myocardial contractility helping to conserve energy or decrease demand.
3. After an MI, a high level of catecholamine's irritates the heart causing an imbalance in supply and demand, which can lead to life-threatening dysrhythmias. Beta-blockers block the harmful effects of catecholamine's improving survival after an MI.
4. The heart spends more time in diastole than in systole.

### **Indications**

1. Most effective in the treatment of exertional angina
2. Antihypertensive treatment

3. Cardiac dysrhythmias
4. Cardio-protective effects following an MI
5. Approved for migraine headaches, essential tremors and tachycardia caused by stage fright.

### **Adverse Effects**

Adverse effects include decreased in heart rate, cardiac output and cardiac contractility. Therefore, bradycardia, hypotension and AV block. In the CNS, it can cause dizziness, fatigue, depression, and lethargy. In diabetic patients, beta-blockers can cause hyperglycemia and/or hypoglycemia as well as masks the signs and symptoms of hypoglycemia and the patients might not be able to tell when exactly the sugar is too low.

### **Calcium Channel Blockers**

CCBs that are used for the treatment of chronic stable angina include amlodipine (Norvasc), diltiazem (Cardizem), nicardipine (Cardene), nifedipine (Procardia), and verapamil.

### **Mechanism of Action**

Prevents calcium from entering the muscle of the heart to prevent muscle contraction and promote muscle relaxation. When the muscle relaxes, it causes the blood vessels to dilate and therefore increases the blood flow to the ischemic heart, which in turn increases the oxygen supply and helps shift the supply/demand ratio back to normal. CCBs cause peripheral arterial vasodilation decreasing systemic vascular resistance and decreasing the workload of the heart, which leads to decrease in myocardial oxygen demand.

### **Indications**

First-line treatment for:

1. Angina
2. Hypertension

### 3. Supraventricular tachycardia

Also, used to treat coronary artery spasms (prinzmetal angina) as well as the short-term management of atrial fibrillation, atrial flutter, migraine headaches, and Raynaud's disease.

#### **Adverse Effects**

Limited adverse effects and those are related to overexpression of therapeutic effects including hypotension, palpitations, tachycardia or bradycardia, constipation, nausea, dyspnea.