



Why Angina Pectoris? Brief Dialogue on Etiopathogenesis & Management: A Petite Explanation to the Diploma Students

Chanda Malviya¹, Priyanka Jain², Tina Malviya³

¹Assistant Professor, VNS Group of Institutions, Faculty of Pharmacy, Bhopal (MP) & PhD scholar at Lovely Professional University, Phagwara, Panjab

²Assistant Professor, VNS Group of Institutions, Faculty of Pharmacy, Bhopal (MP)

³PG scholar, VNS Group of Institutions, Faculty of Pharmacy, Bhopal (MP)

Email id: chanda.malviya98@gmail.com, priyankark2295@gmail.com, tinamalviya542@gmail.com

DOI : <https://doi.org/10.55248/gengpi.5.1024.3001>

ABSTRACT:

Unhealthy lifestyle and poor physical fitness are the most relevant cause of the generation of cardiac difficulties. Often, elevated cholesterol levels increase the risk of myocardial ischemia, leading to the dysfunction of heart cells due to a lack of oxygen (O₂) and vital nutrients. Angina pectoris (AP) becoming a threat for hyperlipidemic, cardiac and anemic patients. Thrombogenesis or embolus formation worsens the situation more due to blockage or restriction in the blood supply, which increases the O₂ demand of cardiac cells needed. This type of anginal pain patients may experience at the time of resting stage. The anginal pain can be managed by using available pharmacological or non-pharmacological therapy. The drugs follow numerous mechanisms to avoid the pain signal eg. Drugs available for vasodilation, include some beta-blockers, Ca⁺⁺ channel blockers, and K⁺ channel openers. This presented review aims to deliver brief knowledge of the generation of AP and its drug management.

Keywords: Chest pain, etiopathogenesis, treatment, management, drug action

1.1 Introduction

The term AP is a disequilibrium between myocardial blood supply and oxygen demand. The obstruction in the blood supply to the heart; experiences severe chest pain in the middle or left side of the chest. AP became one of the root causes of the death due to cardiac dysfunction. The AP pain can be distributed according to the period of pain signals. Unstable angina has more intensity of pain in comparison to stable angina, which can be experienced for longer periods by the affected person.

1.2 Types

Stable angina: Generally occurs at the time of physical activity and stress. This circumstance shows an insufficient supply of blood in the heart muscles that are needed for their proper function due to the contraction of arteries.

Unstable angina: It is a very detrimental and life-threatening kind of angina that occurs in resting conditions. It occurs due to an embolus in the coronary artery which resists blood flow into the heart muscles. The duration of the pain could be 20 minutes.

Prinzmetal angina (Variant): Never affects coronary arteries. This is a rare type of pain not occur after a physical workload. Affects women at the time of rest due to vasospasm of small arteries.

Microvascular angina: This type of angina affects small coronary arteries. The blockage of these arteries causes improper blood supply which is a reason for chest pain.

Refractory Angina: Frequent chest pain due to an ischemic condition, not improved by pharmacological interventions.

1.3 Symptoms

1. Pain in the middle of chest and spread to left arm, back and neck
2. Chest discomfort like; (heaviness, squeezing, burning, or choking sensation)

3. Feeling of pressure
4. Tightness in chest
5. Burning
6. Dizziness
7. Shortness of breath
8. Nausea

1.4 Etiology/Causes/Risk Factors

1. Age
2. High blood pressure
3. Diabetes
4. Unhealthy cholesterol level
5. Anemia
6. Extreme cold
7. Smoking
8. Lack of physical exercise
9. Obesity
10. Excessive consumption of sodium in the diet
11. Excessive consumption of alcohol
12. Family history of coronary heart disease

1.5 Pathogenesis

Formation of thrombus and lipid deposition in the artery obstructing the O_2 supply develop ischemia and lactic acid formation which generate signals of pain.

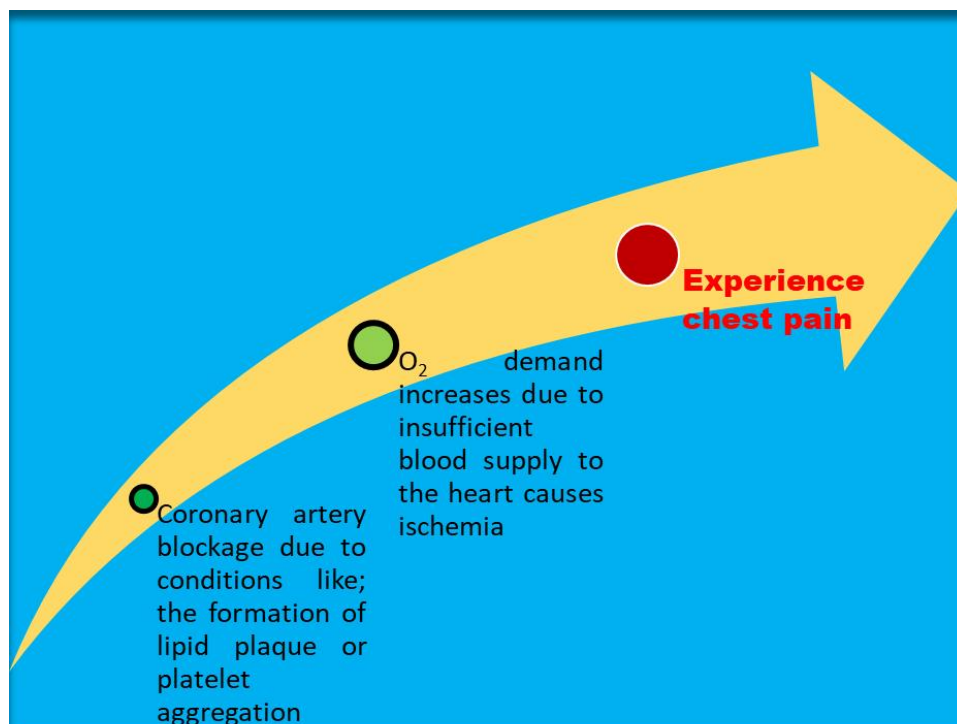


Fig. 1 Development of angina pain due to ischemic condition

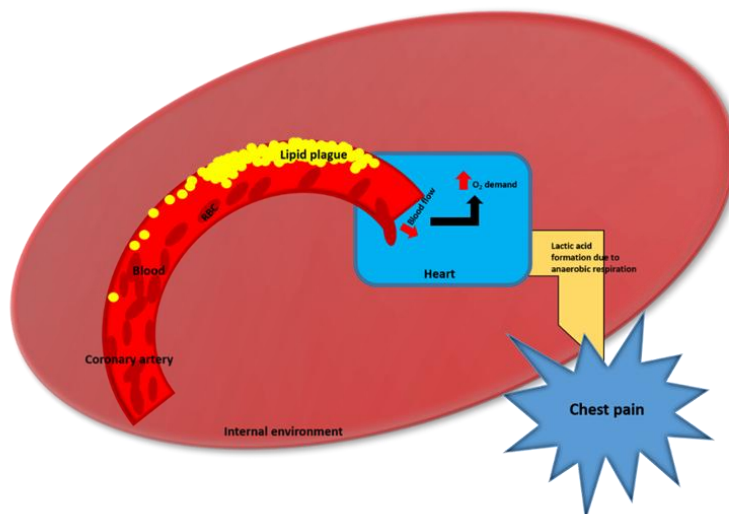


Fig. 2 Coronary artery blockage due to lipid molecule accumulation restricts blood flow and develops pain signals.

1.6 Prevention

1. Maintain unhealthy cholesterol level (limited consumption of fats, salt, and sugar)
2. Improve physical activity
3. Quit smoking
4. Healthy weight
5. Avoid stress

1.7 Diagnosis Parameters

1. Electrocardiogram
2. Chest X-ray
3. Computerized tomography
4. Angiography
5. Echocardiogram

1.8 Pharmacological Treatment

Those drugs improve the blood flow toward the heart by different mechanisms called anti-anginal drugs.

Table: 1 Classification of drug, brand name and their mechanism

S.No.	Classes	Drugs	Brand name	Mechanism
1.	Nitrates	Short acting : <ul style="list-style-type: none"> ▪ Glyceryl trinitrate ▪ Isosorbide dinitrate Long acting : <ul style="list-style-type: none"> ▪ Isosorbide mononitrate 	Rectogesic Isordil Imdur	Vasodilation

		<ul style="list-style-type: none"> ▪ Isosorbide dinitrate ▪ Erythritol tetranitrate ▪ Pentaerythritol tetranitrate 	Sorbitrate Cardilate Duotrate	
2.	Beta-blockers	<ul style="list-style-type: none"> ▪ Propranolol ▪ Metoprolol ▪ Atenolol ▪ Bisoprolol ▪ Esmolol ▪ Nebivolol 	Inderal Lopressor Tenolol Concor Brevibloc Bystolic	Block β_1 receptor
3.	Ca ⁺⁺ channel blockers	<ul style="list-style-type: none"> ▪ Verapamil ▪ Amlodipine ▪ Felodipine ▪ Isradipine ▪ Nicardipine ▪ Nitrendipine ▪ Nifedipine ▪ Nisoldipine ▪ Diltiazem 	Calan, Veracal Norvasc Felogard-5, DynaCirc Nicardilex Nitrepin-10 Nifotab-10 Nisolcare Cardizem	Block influx of Ca ⁺⁺ ions
4.	Potassium channel opener	<ul style="list-style-type: none"> ▪ Nicorandil 	Nicomax-5	Efflux of K ⁺ ions
5.	Anti-anginal drugs	<ul style="list-style-type: none"> ▪ Trimetazidine ▪ Ranolazine ▪ Ivabradine ▪ Oxycodrine ▪ Dipyridamole 	Trimacontin Ranolite Ivabeat-5 Adexor Persantine	Inhibit β - oxidation of free fatty acid Decrease action of sodium and Ca ⁺⁺ Reduce and regulate heart rate Vasodilator Inhibit the function of phosphodiesterase and adenosine deaminase.

1.9 Mechanism of action

- Nitrates:

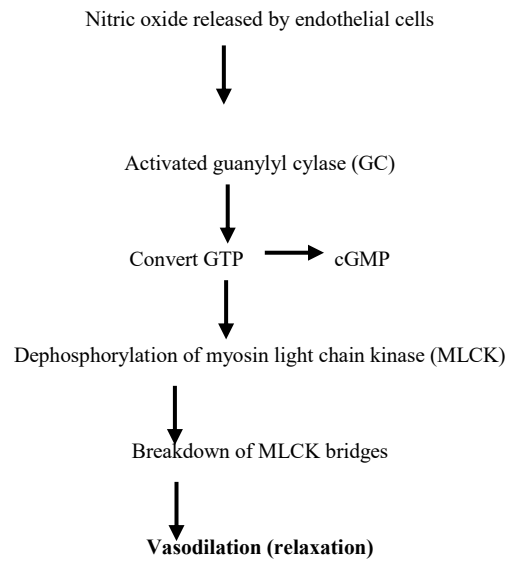


Fig. 3 Flow diagram of nitrates action

1.10 Non-pharmacological treatment

1. Regular exercise
2. Healthy diet
3. Medical counseling

1.11 Side effects of drugs

S.No.	Drugs	Side effects
01	Nitrates	<ol style="list-style-type: none"> 1. Flushing of face and neck 2. Headache 3. Dizziness 4. Sweating 5. Hypotension 6. Upset stomach
02	Beta-blocker	<ol style="list-style-type: none"> 1. Slow heartbeat 2. Cold hand 3. Headache 4. Weakness 5. Wheezing 6. Dizziness
03	Calcium channel blocker: Verapamil	<ol style="list-style-type: none"> 1. Nausea, 2. Bradycardia

	Nifedipine	3. constipation 1. Palpitation 2. Hypotension 3. Headache 4. Drowsiness 5. Ankle edema
04	Ranolazine	1. Dizziness 2. Swelling of face, arm and legs 3. Headache 4. Weight gain 5. Spinning movement

Table: 2 Categories side effects of drugs

Conclusion:

Unstable angina is the one of the detrimental situation which needed pharmacological interventions whereas, stable angina can be also manage by lifestyle changes. To reduce mortality due to ischemic heart population need to understand and improve their regular activities to keep themselves healthy.

References:

- <https://www.hopkinsmedicine.org/health/conditions-and-diseases/angina-pectoris#:~:text=Angina%20pectoris%20is%20chest%20pain,it%20can%20have%20other%20causes.>
- <https://www.mayoclinic.org/diseases-conditions/angina/symptoms-causes/syc-20369373>
- Manfredi R, Verdoia M, Compagnucci P, Barbarossa A, Stronati G, Casella M, Dello Russo A, Guerra F, Ciliberti G. Angina in 2022: Current Perspectives. *J Clin Med.* 2022 Nov 22;11(23):6891. doi: 10.3390/jcm11236891.
- Hermiz C, Sedhai YR. Angina. [Updated 2023 Jun 6]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK557672>.
- Chrusciel P, Rysz J, Banach M. Defining the role of trimetazidine in the treatment of cardiovascular disorders: some insights on its role in heart failure and peripheral artery disease. *Drugs.* 2014 Jun;74(9):971-80. doi: 10.1007/s40265-014-0233-5.
- Reed M, Kerndt CC, Gopal S, et al. Ranolazine. [Updated 2024 Feb 28]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK507828>.
- Tripathi KD. *Essential of Medical Pharmacology.* Jaypee brother medical publisher LTD. 2013. 7th edition. 539
- Reed M, Kerndt CC, Nicolas D. Ivabradine. [Updated 2023 May 16]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK507783>.
- Chichkanov GG, Chumberidze VB. K mekhanizmu antianginal'nogo deistviia oksifedrina [Mechanism of the antianginal action of oxyfedrine]. *Farmakol Toksikol.* 1977 May-Jun;40(3):302-6.
- Otsuki Y, Yamasaki J, Suina K, Okazaki S, Koike N, Saya H, Nagano O. Vasodilator oxyfedrine inhibits aldehyde metabolism and thereby sensitizes cancer cells to xCT-targeted therapy. *Cancer Sci.* 2020 Jan;111(1):127-136. doi: 10.1111/cas.14224. Epub 2019 Nov 22.
- Kerndt CC, Nagalli S. Dipyridamole. [Updated 2023 Jul 3]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK554455>.
- Kim KH, Kerndt CC, Adnan G, et al. Nitroglycerin. [Updated 2023 Jul 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK482382>.

-
13. Torfgård KE, Ahlner J. Mechanisms of action of nitrates. *Cardiovasc Drugs Ther.* 1994 Oct;8(5):701-17. doi: 10.1007/BF00877117.
 14. Shahrokhi M, Gupta V. Propranolol. [Updated 2023 May 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK557801>.
 15. Rakhimov K, Gori T. Non-pharmacological Treatment of Refractory Angina and Microvascular Angina. *Biomedicines.* 2020 Aug 13;8(8):285. doi: 10.3390/biomedicines8080285.