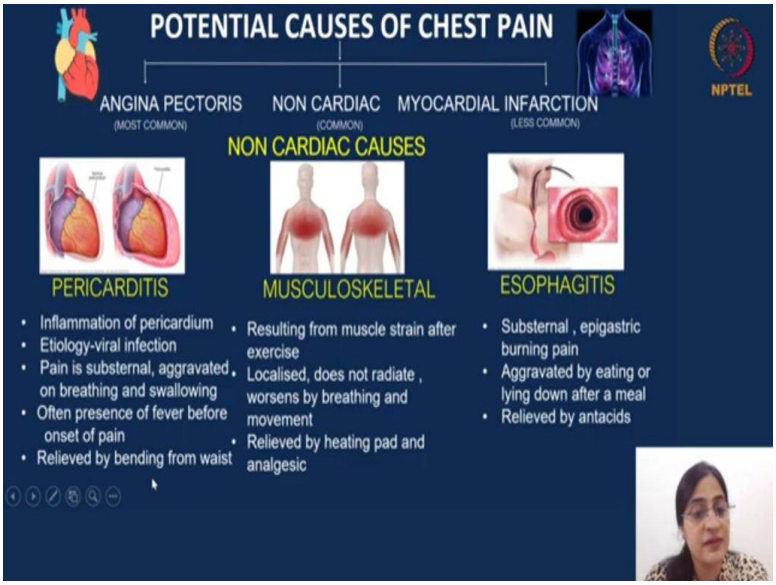
**Management of Medical Emergencies in Dental Practice**
**Professor Doctor Charu Girotra**
**D.Y. Patil University School of Dentistry**
**Lecture 10**
**Chest Pain of Cardiac Origin-Myocardial Infarction and Anigina Pectoris-Part 1**

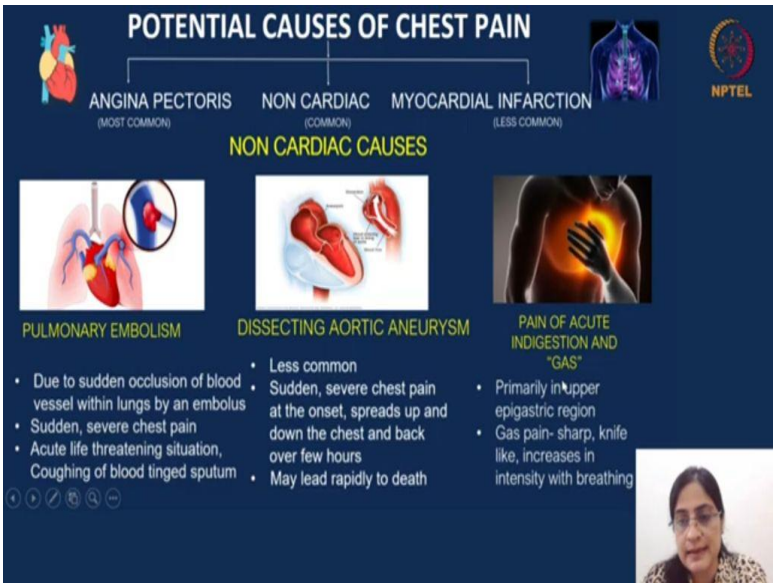
Hello everyone, I welcome you all to the third week of course on medical emergencies. In this week we will be focusing on chest pain of cardiac origin, mainly angina pectoris and myocardial infarction.

(Refer Slide Time: 00:26)


All of us experience some kind of chest pain, but mostly these, this chest pain is unrelated to ischemic heart diseases, and it is not life threatening. Now as a clinician, we must be able to differentiate the cardiac chest pain from non-cardiac chest pain. When we talk about cardiac chest pain, most commonly is the ischemic heart disease the reason of it. And in ischemic heart diseases, angina pectoris is the most common, while myocardial infarction is less common.

(Refer Slide Time: 00:55)




So, let us have a look at the key features of chest pain associated with non cardiac causes. The most common is musculoskeletal results from a muscle strain after physical exercise would be cardio exercise or muscle training in gym; it is localised, does not radiate, it is worsened by breathing and movement. It is relieved by heating pad analgesic.

Now, second common factor is esophagitis. Again, the pain is substernal epigastric; it is a burning kind of pain aggravated by eating or lying down after a meal, relived by antacids. In case of pericarditis, the pain is due to inflammation of (pericard) pericardium.

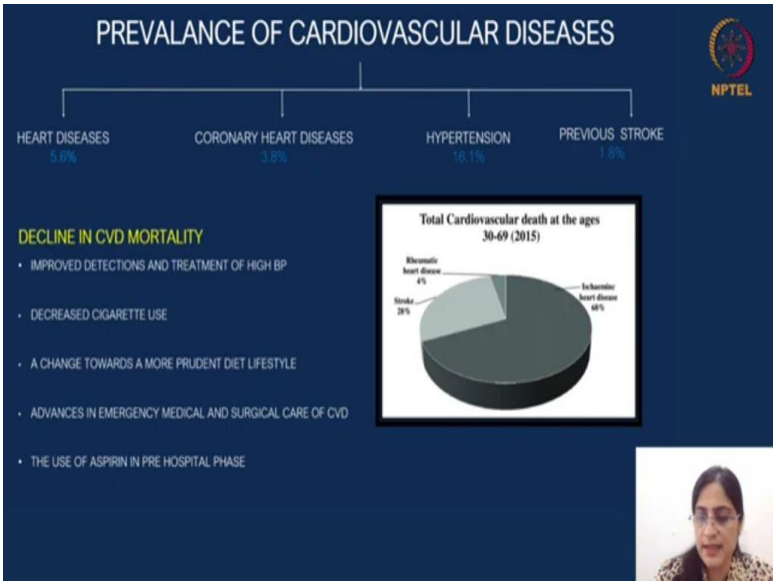
The etiology is viral infection, the location of pain is again substernal, aggravated on breathing and swallowing. There is often presence of fever before the onset of pain and it is relieved by bending from waste. Another common non cardiac cause is pain of acute indigestion and gas;

it is primarily located in upper epigastric region, the pain is sharp, knife like, increases intensity with breathing and relieved by antacids.

Now, less common causes include dissecting aortic aneurysm and pulmonary embolism. What do we mean by dissecting aortic aneurysm? Aneurysm is abnormal dilatation of aorta, here you can see it. And dissecting means there is a interior in the intima layer of aorta; it is less common.

The pain is sudden; it is severe at the onset, spreads up and down the chest and back over few hours, may lead to death. Less common another non cardiac cause is pulmonary embolism due to sudden occlusion of blood vessel within lungs by an embolus, which is thrown out or broken off from deep veins. The pain is sudden, it is severe. It is an acute life threatening situation and accompanied with coughing of blood tinged sputum.

(Refer Slide Time: 02:50)




Now, after the differential diagnosis of chest pain, let us discuss the prevalence of ischemic heart diseases. Ischemic heart diseases are also known by the term coronary heart disease or coronary artery diseases. Now, these constitute 3.8 percent of cardiovascular diseases; this is the factor which is in Asia.

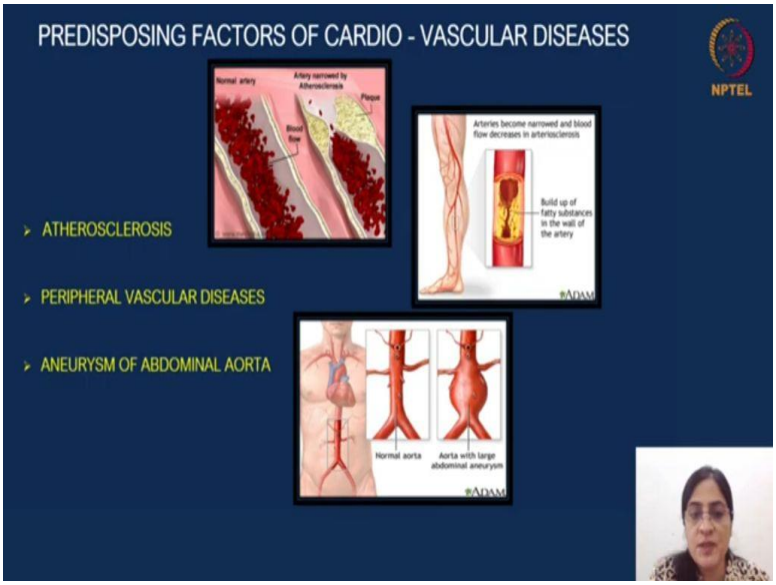
If we look at India statistics, the cardiovascular deaths are most commonly caused by ischemic heart diseases that constitutes 68 percent of cardiovascular diseases. But, still we have declined in mortality due to cardiovascular diseases; and the reasons are improved detections and treatment of high blood pressure, decreased cigarette use; a change towards a more prudent diet lifestyle.

There are advances in emergency medical and surgical care of cardiovascular diseases; also the use of aspirin in pre-hospital phase. Aspirin use can be therapeutic or prophylactic; prophylactic use in chronic hypertensive patients, or diabetic patients, or in old age; and therapeutic use can be in cardiovascular diseases.

(Refer Slide Time: 03:57)


Why do the patients suffering from ischemic heart disease represent a great risk during routine dental treatment? Because, these patients would be ambulatory; that means walking around normally doing their routine daily procedures. These patients would be asymptomatic and some of them would not know their cardiovascular status. So, any dental procedure which can increase the stress cause an emotional or physical stress to the patient, and increase the workload of patient's cardiovascular system is potentially dangerous.

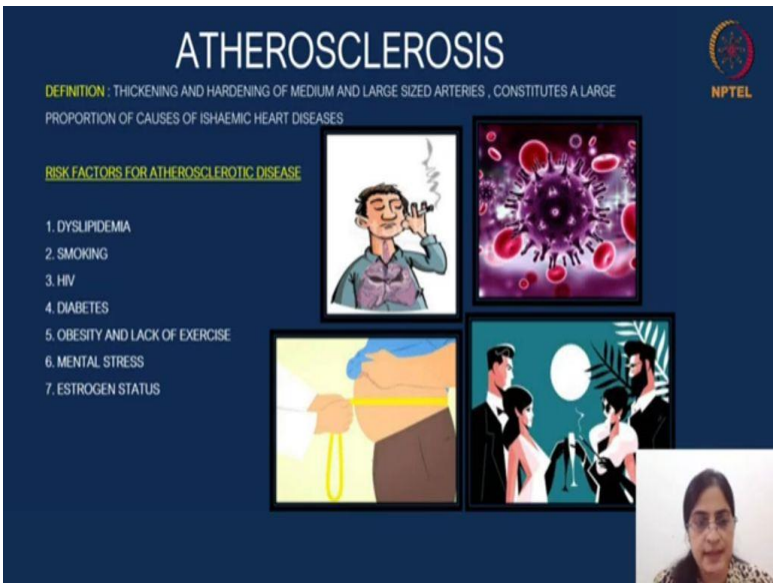
(Refer Slide Time: 04:27)




Now, when we look at the cardiovascular diseases, we must know the predisposing factors leading to cardiovascular diseases. And there there are three main factors: atherosclerosis, peripheral vascular diseases, and aneurysm of abdominal aorta. Atherosclerosis is the single main factor constituting 70 percent of ischemic heart diseases, and even peripheral vascular diseases.

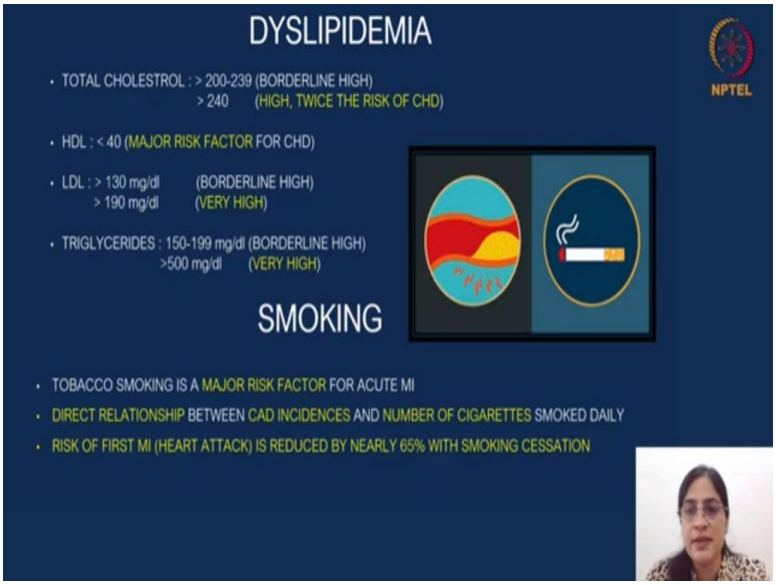
When we talk about peripheral vascular disease, again, arteriosclerosis is present. And it leads to the narrowing of the vessels and reduce blood supply to the extremities, which again is a threat to cardiovascular system. And aneurysm of abdominal aorta when it ruptures, it can lead to myocardial infarction; it can lead to stroke and even death.

(Refer Slide Time: 05:12)



So, let us discuss atherosclerosis little bit in detail. How do you define it? It is thickening and hardening of medium and large sized arteries. Now, what are the risk factors which are responsible for atherosclerotic disease? Dyslipidemia, that means abnormal lipid profile, smoking, HIV, diabetes, obesity and lack of exercise, mental stress, and estrogen status. Let us pick them one by one.

(Refer Slide Time: 05:40)




Now, Dyslipidemia, why it is relevant to us? Because when we are looking at the reports of the patient, we must know what are the abnormal lipid parameters and what is the risk to the cardiovascular system. So, if the total cholesterol is more than 240, it is very high; and that particular patient will have twice the risk of coronary artery disease.

HDL is considered to be a good cholesterol. So, if it is less than 40, it constitute major risk factor for coronary artery diseases. LDL which is low density lipoproteins is considered to be a bad cholesterol. And if it is more than 130, it is considered to be high; and a threat for ischemic heart diseases. Triglycerides; more than 200 are borderline high, while more than 500 is very high factor.

Now smoking; smoking is also considered a major risk factor for acute myocardial infarction. And anything when we explain to the patient and we have to make the patient aware of the bad habits, we must have a literature support. And the evidence from literature says that there is a direct relationship between incidence of coronary artery diseases, and the number of cigarettes smoked daily. And the very risk of first MI is reduced by nearly 65 percent, if the patient stops smoking.

(Refer Slide Time: 06:57)


Hypertension; now again, the literature support any in every seven millimeter increase of diastolic blood pressure, increases the risk of coronary artery disease by 30 percent approximately. And there is a pharmacological reduction of diastolic blood pressure by five millimeters, which can lead to reduction of risk of coronary artery disease by 14 percent. Now, what happens in a normal patient in a normal scenario, when the patient does not have high blood pressure? There is an inflow and efflux. You can see influx and efflux of lipids in and out of the blood vessel.

Now, if there are changes in the blood pressure, the intimal layer of the blood vessel undergoes changes; it goes for proliferation, so there is influx of lipids happening pre-dominantly rather than efflux. Now, this lipid deposition over a period of time, it narrows the vessels and reduces the blood supply to that area.

Now, whatever damage is done over a period of time, we cannot undo it; but controlling the blood pressure can reduce or slow down the process of atherosclerosis. So, it is very important for us to check the vital signs, pre-op and post-op whenever we are doing the routine dental procedure. Now, insulin resistance and diabetes. Diabetics also have increased rate of cardiovascular events, and even insulin resistance leads to type-2 diabetes, and it can promote atherosclerosis.

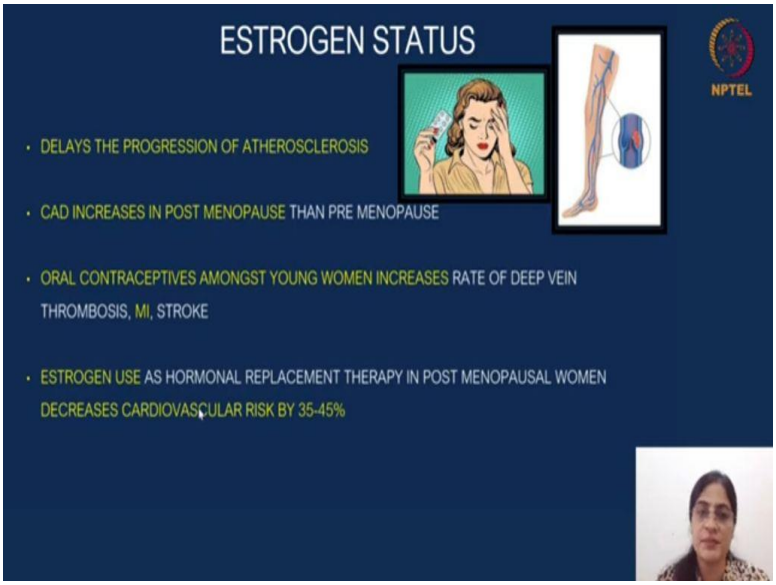
(Refer Slide Time: 08:28)


Lack of exercise and obesity will increase the myocardial oxygen demand; and this will increase the coronary risk. So, regular physical exercise will reduce the weight, it will decrease the myocardial oxygen demand. It will have favorable effects on blood pressure, lipid profiles, improved glucose tolerance; and thereby decreasing the risk of coronary artery disease.

Coming to the mental status of the patient. When we talk about the mental status, we are talking about the patients who are over anxious, or they have a phobia towards dental treatment. So, what happens in such patients if the patients are very anxious? They will have release of adrenaline, which is multiple folds.

So, this adrenergic stimulation will increase the myocardial oxygen demand and aggravate the myocardial ischemia. Also, it can cause coronary vasoconstriction in already for atherosclerotic coronary arteries; and it can also alter thrombosis and coagulation, and favour the clot formation. So, it is important that the dental clinic environment, we have to make it least stressful for the fearful patients.

(Refer Slide Time: 09:39)




Lastly is the estrogen status. As per the literature estrogen is a good hormone; it delay the progression of atherosclerosis. Why the literature says so, because the incidence of coronary artery diseases is more in postmenopausal woman than pre-menopause. Also, the women who are on long term use of oral contraceptives have higher risk of deep vein thrombosis, myocardial infarction and even stroke.

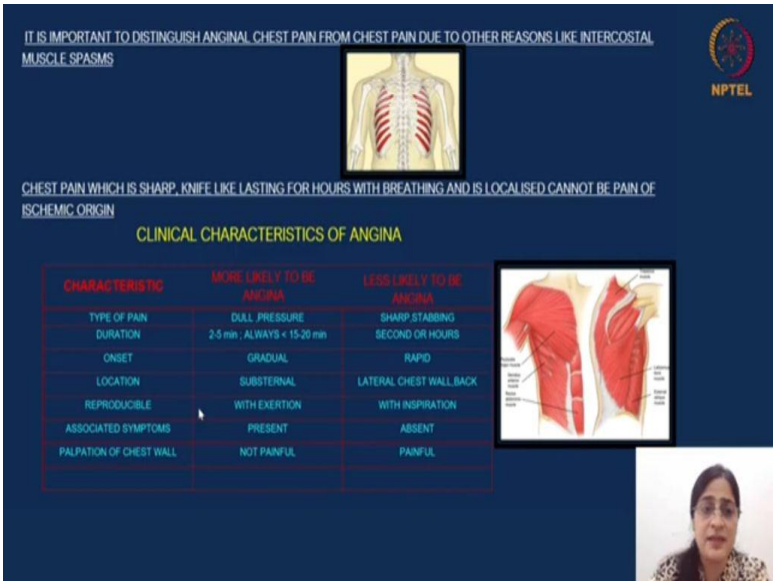
(Refer Slide Time: 10:06)


So, after discussing the predisposing factors, let us come to angina pectoris, which is the most common ischemic heart disease; angina and pectoris both are Latin words. Angina meaning spasmodic, cramp like or choking feeling, or a suffocating kind of pain by pectoris means chest.

This term was first ever used by Dr. William Heberden to distinguish the strangling feeling of angina from DOLOR, which means pain.

Now, how do we define angina pectoris? It is a characteristic thoracic pain, substernal, precipitated by exercise, or emotion or a heavy meal, relieved by vasodilator drugs, or a few minutes rest; and it is usually a result of a moderate inadequacy of coronary circulation. Now, how do the patients describe this pain? They describe this pain as crushing, heavy, suffocating, constricting, squeezing and dull or aching discomfort.

(Refer Slide Time: 11:11)




So, we need to differentiate a anginal pain from a non-anginal pain. So, what are the characteristics of the pain which are more likely to be angina? The pain would be dull and pressure kind of feeling in angina. And the pain which is sharp and stabbing is less likely to be angina. The duration of pain which is 2 to 5 minutes; and always less than 15 to 20 minutes can be angina. But, the duration of pain which is seconds or going for hours together can, is less likely to be angina.

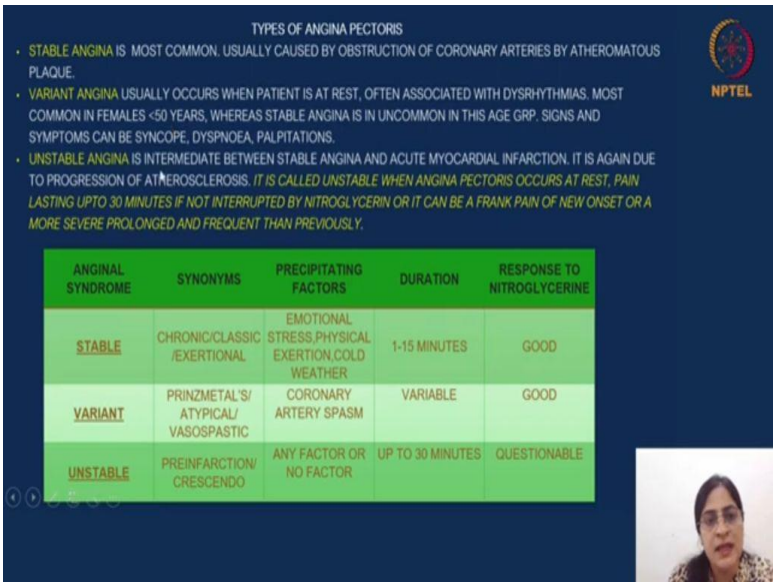
The onset of angina pain is gradual, rather than rapid. The location is substernal rather than lateral chest wall or back, it is aggravated with exertion. It is usually happening at the time of exertion, rather than with inspiration. Associated symptoms can be dyspnoea, can be syncope; it can be tachycardia, while it is absent in other causes. And palpation of chest wall, when you palpate the chest wall, it is non-tender if it is an angina pain as compared to a non-anginal pain.

(Refer Slide Time: 12:16)


So, what are the factors which are leading to angina pectoris? It can be precipitated by four E's; we call it as the triggers. The four E include emotion, by emotion, we mean anxiety or excitement; exercise that is physical activity; exposure to cold or hot and humid climate; eating basically a large meal. So, these four E's are responsible for increasing the myocardial oxygen demand; and which the coronary arteries are unable to supply.

So, there is basically disparity between the demand and supply of oxygen which leads to angina. What can be the other factors? The other factors can be caffeine ingestion, or acute fever, anemia, thyrotoxicosis, cigarette smoking, smog, or even high altitudes.

(Refer Slide Time: 13:06)



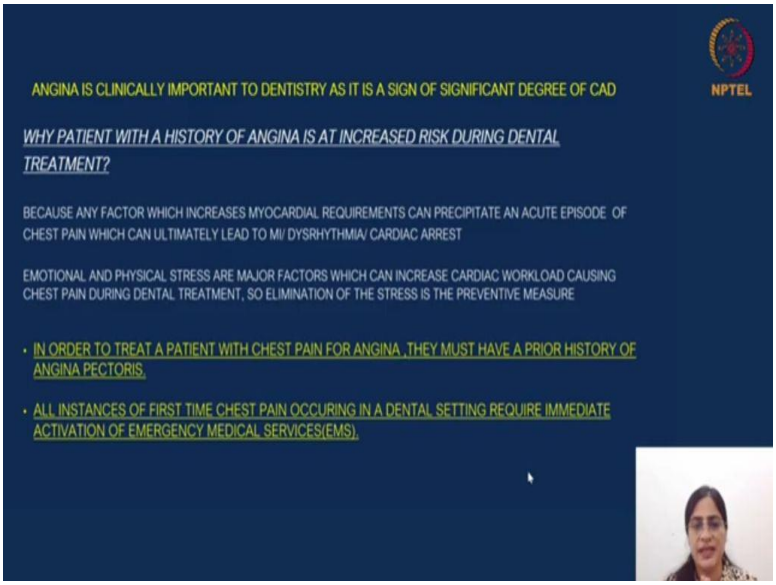
So, what are the types of angina pectoris? We have basically three types of angina pectoris. Stable angina is the most common; and it is usually caused by obstruction of coronary arteries by atherosclerosis. And the synonyms for stable angina are chronic, classic, exertional. The precipitated factors include the four E's, the duration could be few minutes less than 15 minutes; and the response to nitroglycerin is good in case of stable angina.

Now, let us come to variant angina. Variant angina is also called as prinzmetal's or atypical or vasospastic angina. It usually occurs when the patient is at rest; associated with dysrhythmias, most common in females less than 50 years of age. And signs and symptoms can be syncope, dyspnoea palpitations, the precipitating factor can be coronary artery spasm. Duration is variable and the response to nitroglycerin is good in this case also.

Unstable angina; unstable angina is the intermediate between the stable angina and acute myocardial infarction. It is again due to the progression of atherosclerosis; it is also called as pre-infarction or crescendo. Any factor or no factor is responsible for causing unstable angina. Now, in what scenarios it is called unstable? It is called unstable when angina pectoris occurs at rest.

Because the stable angina most commonly is happening with the exercise; or in case of pain which is lasting up to 30 minutes; that means more than 15 minutes if not interrupted by nitroglycerin. Or it can be a sudden acute pain of new onset that pain has never happened before; or a more severe, prolonged and frequent than previous pain that can also be an unstable angina pain. So, more than 15 minutes up to 30 minutes is the duration; and response to nitroglycerin will be questionable in case of unstable angina.

(Refer Slide Time: 15:10)

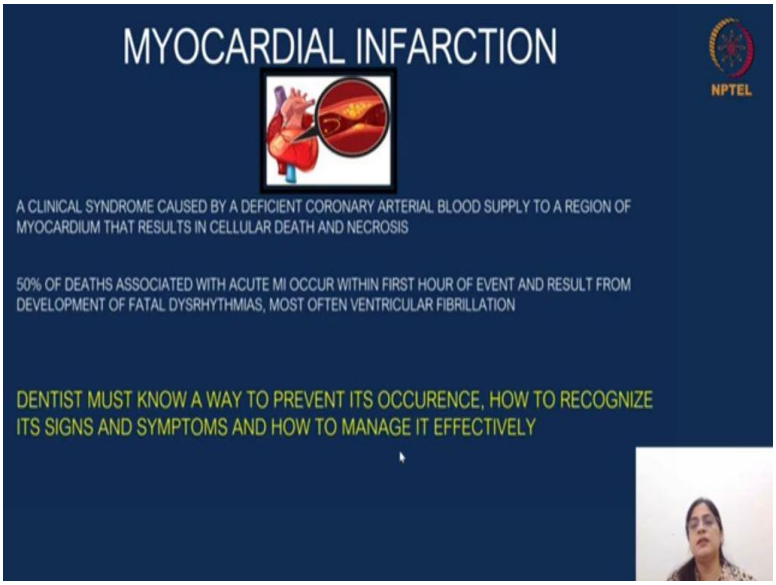



Why a patient with a history of angina is important to us is clinically relevant to us; and why he or she is at increased risk during dental treatment? Because any factor, basically here the factors when we are doing a routine dental procedure or any dental procedure, we are talking about the factors like emotional and the physical factors.

These are the major factors which can increase the myocardial oxygen demand, which can increase the cardiac workload causing chest pain during dental treatment. So, elimination of this emotional and physical stress is what we need to achieve and we want to prevent any kind of anginal attack in dental chair.

So, in case the dent in the dental chair, the anginal pain happens. We are supposed to treat a patient with chest pain for angina, only if the patient has given you a prior history of angina pectoris. If it is a first time chest pain in a dental setting, then it requires immediate activation of emergency medical services.

(Refer Slide Time: 16:18)

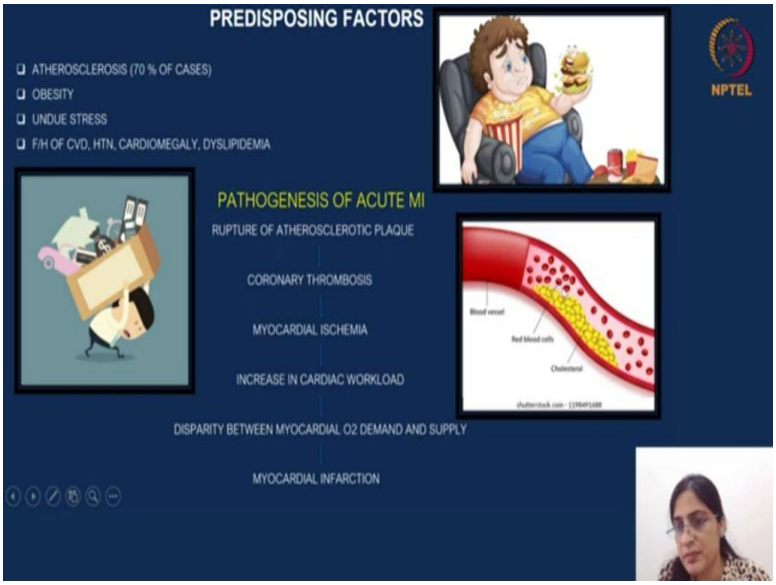



So, second common ischemic heart disease we are going to understand is the myocardial infarction. What is myocardial infarction? It is a clinical syndrome caused by a deficient coronary arterial blood supply to a region of myocardium that results in cellular death and necrosis. So, that is how you differentiate the angina pectoris from myocardial infarction. Angina Pectoris is also caused by a deficient coronary arterial blood supply, but there is no necrosis which has happened.

There is no cellular death which has happened; there is definitely decreased blood flow. So that is causing more workload on the heart and we have an anginal pain. But in case there is a complete blockage of a coronary artery and the area of the myocardium undergoes necrosis, there is a cell death; then it becomes myocardial infarction.

Now, 50 percent of the deaths associated with acute myocardial infarction occur within the first hour of the event. So, it is very important that that first hour is very important for us to transport the patient to the hospital for attention, and it results from development of complications of myocardial infarction which is mainly dysrhythmias, and most often ventricular fibrillation. So, as a dentist, we must know a way to prevent its occurrence first of all, and we must know how to recognize the signs and symptoms, so that we can manage it effectively.

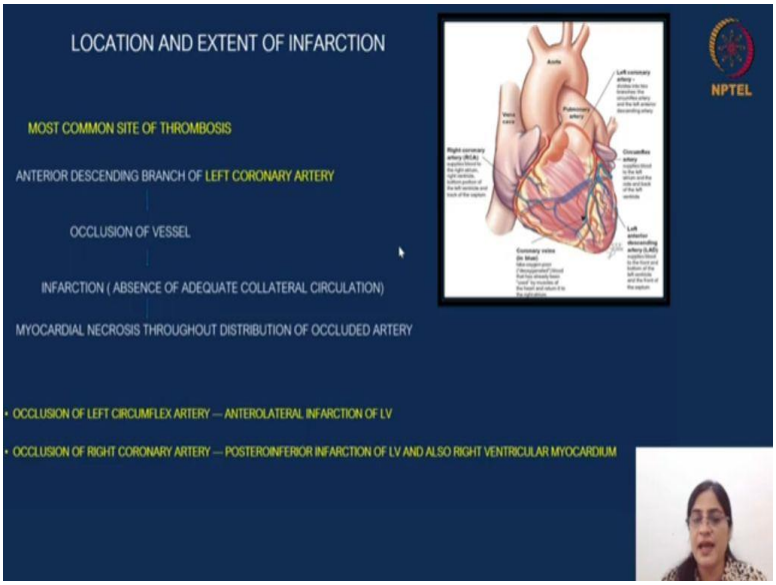
(Refer Slide Time: 17:49)




So, what are the predisposing factors? They are seen as angina pectoris, atherosclerosis, obesity, undue stress, family history of cardiovascular diseases, hypertension, cardiomegaly, abnormal lipid profiles. So, what happens in acute myocardial infarction? So there is a atherosclerotic plaque which is formed, and usually this atherosclerotic plaque has lipid depositions; and it is covered by a capsule.

When this capsule is broken, so the platelets are attracted; and there is a platelet plaque formation; a thrombus formation, which eventually gets dislodged. So, coronary thrombosis, the thrombus can get dislodged; and it can block coronary artery leading to myocardial ischemia, there is increase in cardiac workload. And definitely there is a discrepancy between the oxygen demand and supply leading to myocardial infarction.

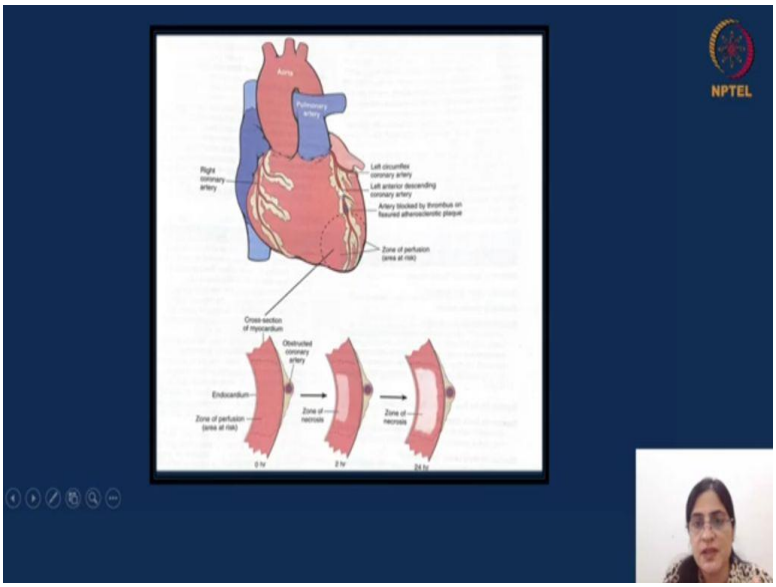
(Refer Slide Time: 18:46)




So, where is most commonly which is the most common vessel which is affected by myocardial infarction? The most common site of thrombosis is the left coronary artery. This is the left coronary artery and its branch is this one, the left anterior descending artery; this is the most common branch which is affected by thrombosis. So there is occlusion of the vessel and leading to infarction. And why the infarction happens because of the absence of the adequate collateral circulation; so, there is myocardial necrosis throughout the distribution of the occluded artery.

Less common vessels are the occlusion of left circumflex artery. So, the anterolateral infarction of left ventricle happens. And if there is an infarction of right in thrombosis of right coronary artery, it will lead to the infarction posteroinferior infarction of the left ventricle; and also the right ventricular myocardium.

(Refer Slide Time: 19:45)

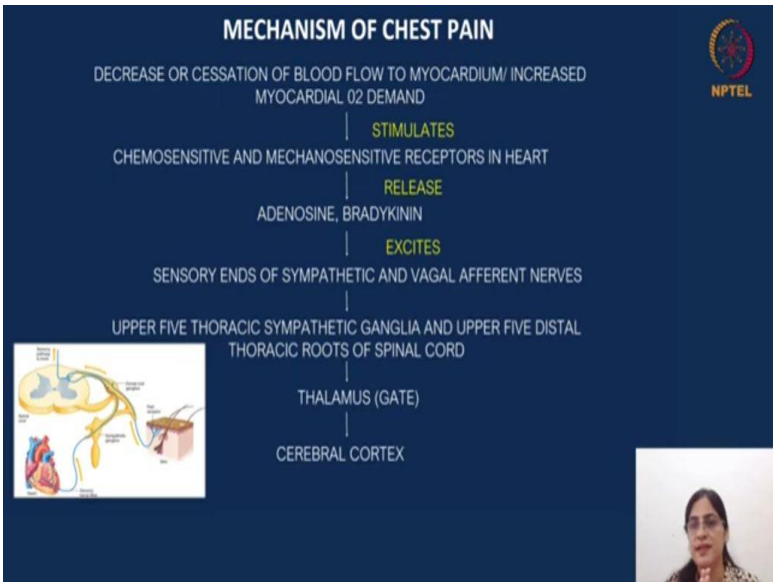




So, what determines the extent of infarction or the area of the myocardial damage, the distribution of occluded vessels? What is the adequacy of collateral circulation at that time? Whether it is one vessel involved or multiple vessels involved and if there was any preexisting infarction?

So, this is a diagrammatic picture which is showing that this is this is the left anterior descending branch of coronary artery, which is completely blocked by the thrombus. And this is the area, the inferior part of the left ventricle, which is affected; and the septum which is affected of the myocardium this, this area is at risk. Now, how important is the time? At zero hour, the this area which is affected of the myocardium is not yet necrosed.

But as there is two hours, which have already gone after the myocardial infarction that starts undergoing necrosis. And the area of necrosis enlarges, area of necrosis enlarges at the end of the 24 hours. So, this is very vital that we transport the patients within that first hour of myocardial infarction.

(Refer Slide Time: 20:58)

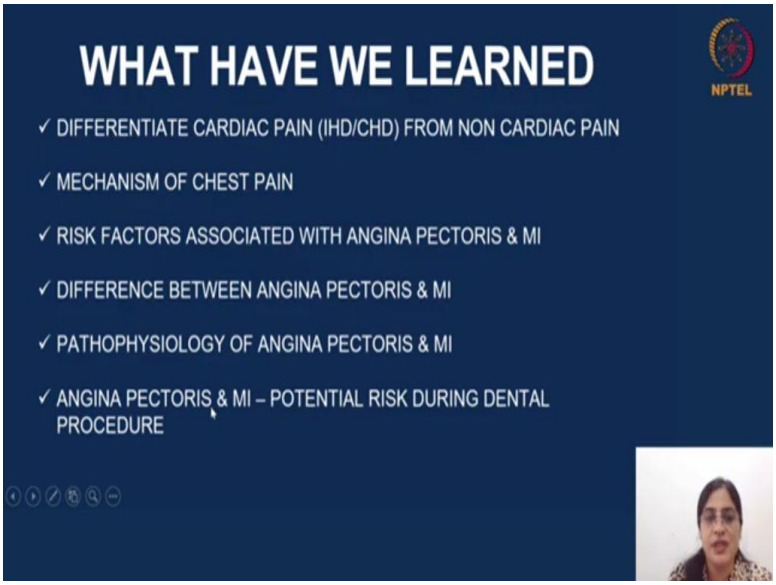



So, after talking about angina pectoris myocardial infarction, now we know what are these diseases? What is the pathophysiology? We must know the pathway of the cardiac chest pain, how the pain originates from myocardium. And once it reaches the cerebral cortex and the patient feels the chest pain.

So, what happens there is a decrease or stoppage of blood flow to the myocardium; or there is increased myocardial oxygen demand. This will stimulate the chemosensitive and mechanosensitive receptors in heart. This will release, these receptors will release adenosine and bradykinin chemical mediators, which will excite the sensory ends of sympathetic and vagal efferent nerves.

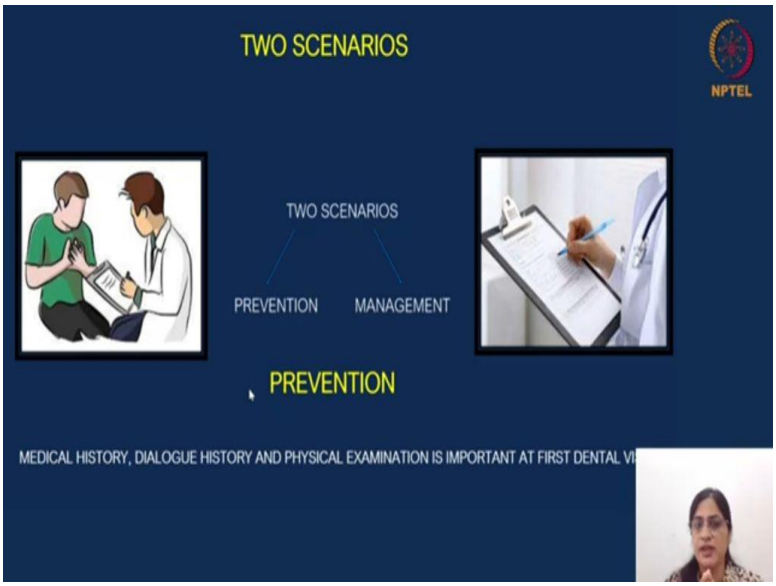
Now, this stimulus further goes to thoracic sympathetic ganglia, and the thoracic roots of spinal cord, which cross the thalamus; which is the gateway to the cerebral cortex and the patient feels chest pain.

(Refer Slide Time: 21:53)




So, what did we learn from the talk of angina pectoris and myocardial infarction till now? We know how to differentiate the cardiac chest pain from ischemic heart diseases or coronary heart diseases from non-cardiac pain. We know the mechanism of pathway of chest pain. We know the risk factors associated with angina pectoris in myocardial infarction, what is the basic difference between the two? What is the pathophysiology and why the patient of angina pectoris and myocardial infraction is a potential risk during dental procedure?

(Refer Slide Time: 22:31)

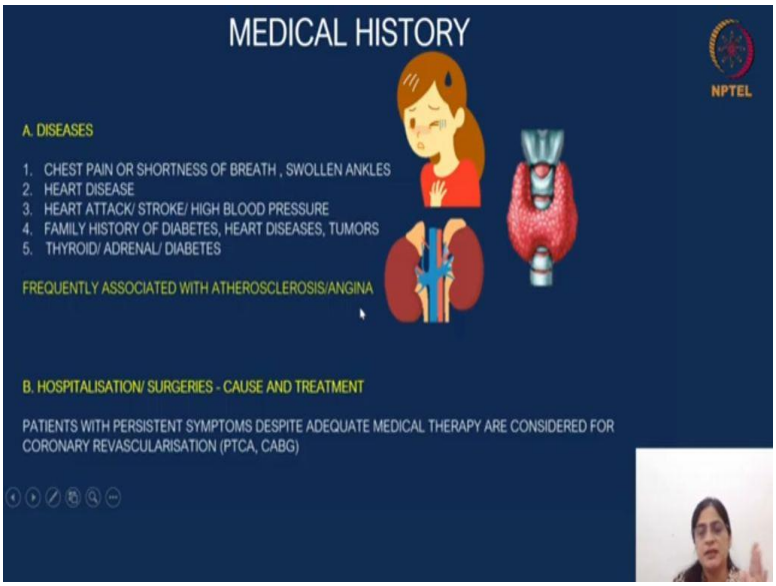



Now, now, we have discussed the pathophysiology; so we have two scenarios in front of us. One is the prevention and the other is management. Under prevention, we have two category

of patients first category of patients who give us the history of angina pectoris. And we do not want any other attack of angina pectoris or myocardial infarction in dental check.

Second category, the patients who are high risk category, but have not experienced any kind of anginal pain or myocardial infarction till date. So, this is the categories of patients we are going to deal with, when we want to prevent any such episode to happen. And in case of management also, we will have certain categories of patients that we will talk about later. So, coming to the prevention what can prevent an episode of thorough medical history, dialogue history and physical examination is very vital at the very first dental visit.

(Refer Slide Time: 23:28)



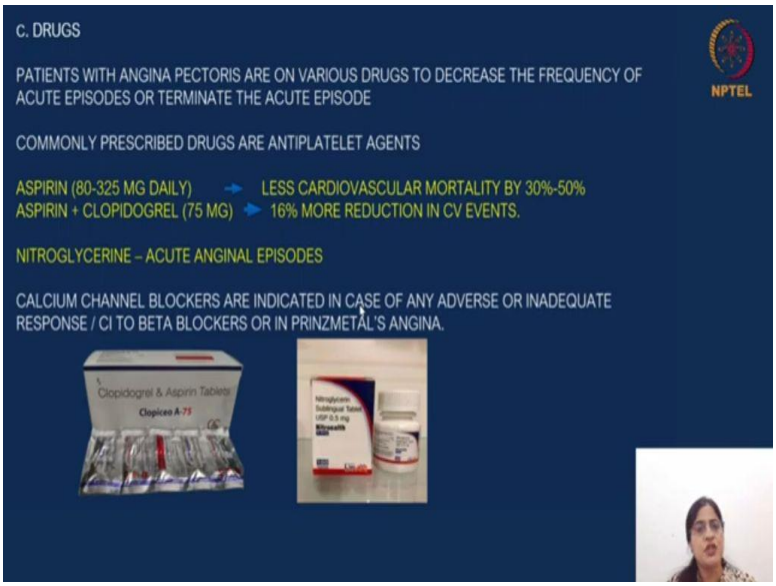

Now, in medical history, what are the diseases which the patient can suffer from and they carry a potential risk of developing a cardiovascular event in dental chair? If the patient has chest pain or shortness of breath, look for swollen ankles. If the patient gives you a history of heart disease or previous heart attack, stroke, high blood pressure, if there is any family history of diabetes, heart diseases, tumors, thyroid, adrenal diabetes; all these endocrinal disorders have to be ruled out.

These are the diseases which are frequently associated with atherosclerosis or angina. Currently, we must keep COVID also in mind, because we have seen in last two years the complication of pericardium due to the COVID. So, there have been patients who have suffered pericarditis and their ejection fraction gone down to 5-10 percent, with the history of corded with the history of COVID.

So, that history also has to be ruled out that it can trigger a cardiovascular event in the dental chair. Ask for the patient if there is a history of previous hospitalizations for any kind of ailments, and what was the cause of the ailment. What was the management done, ask for the any surgeries done; surgeries like there can be a bypass grafting which the patient has underwent, or some angioplasty which the patient has undergone.

So, these are the patients who can have persistent symptoms despite the adequate medical therapy, and they are considered for coronary revascularization. So, they also are a potential risk of developing a cardiovascular event during dental procedure.

(Refer Slide Time: 25:08)




Now, the other way of finding out the medical history is to find out what the patient is having, what kind of medications. Now, the patient usually carries the file or the hospital records, or even the dentist can ask for the hospital records, and go through the records properly. It will give you the detailing of the work that investigations were done, what the patient's suffered from, what drugs the patient has been on.

So, patients with angina pectoris are on various drugs to decrease the frequency of acute episodes or terminate in acute episodes; so, commonly prescribed drugs are antiplatelet agents. It could be just a single antiplatelet agent like aspirin, some 75 to 325 milligrams taken daily.

And it will reduce the cardiovascular mortality by 30 to 50 percent. Or, it can be a dual antiplatelet therapy in which aspirin is given in combination with clopidogrel, usually 75 milligrams. And it has been shown that it causes 16 percent more reduction in cardiovascular

events, added to single dose. And for acute anginal episodes, these patients are told to take nitroglycerin.

Now, there are some patients who would be on calcium channel blockers also, they are indicated in case of any adverse or inadequate response to beta blockers or contraindication to beta blockers, or in case of prinzmetal angina. So, when we talk about the antiplatelet, this is the aspirin or a combination of clopidogrel and an aspirin mostly given to the patients. This is the nitroglycerin tablets.

(Refer Slide Time: 26:50)



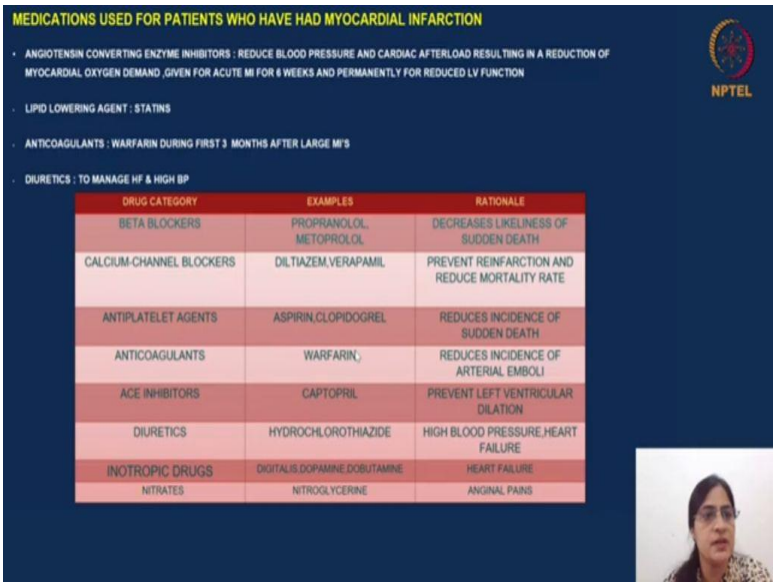
So, aspirin and clopidogrel, I will not get into the brand names of it. Usually most common is the ecosprin; and it can range from 75 to 150 milligrams of clopivas. As a dentist, as a medical practitioner, we must be aware of what newer new drugs are going on, for any kind of

medication which is given in angina pectoris and myocardial infarction; like in antiplatelet therapy some new drugs are introduced. So, it is very important to go through the file of the patient with an even in anticoagulant therapy, there are some new drugs under trial.

(Refer Slide Time: 27:29)


So, these are the basic beta and blockers and the calcium channel blockers, which are given to prevent or treat angina pectoris.

(Refer Slide Time: 27:40)




So, other than these drugs, there are there are more drugs added for the patients who have suffered myocardial infarction. They are the ACE inhibitors, the angiotensin converting enzyme inhibitors, or angiotensin receptor blockers also. What is their purpose? They reduce the blood pressure and cardiac afterload resulting in a reduction of myocardial oxygen demand.

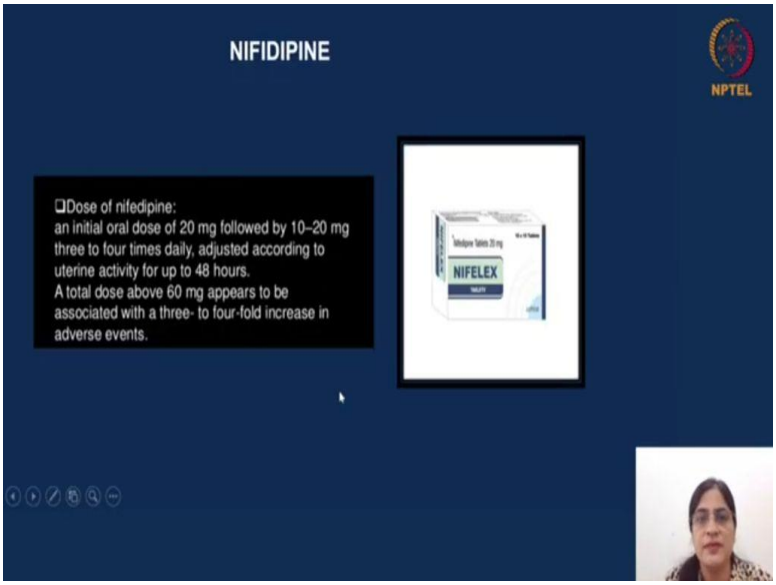
It is usually given for acute myocardial infarction for six weeks at least; and permanently, if there is reduced left ventricular function.

These patients would be on lipid lowering agents also mostly statins. They will be on anticoagulants, at least for first three months after large myocardial infarctions; and the most common anticoagulant we all know is warfarin. These patients can be on diuretics, also like thiazides.

So, this will be helpful to manage a heart failure and high blood pressure. So, these all are the categories of the drugs which the patient of myocardial infarction might be put on for the rationale given here; like beta blockers to decrease the likeliness of sudden death. Calcium-channel blockers will prevent the reinfarction and reduce the mortality rate.

Antiplatelet agents reduce the incidence of sudden death; anticoagulants will reduce the incidence of arterial emboli. ACE inhibitors to prevent any left ventricular dilation; diuretic, diuretics to treat high blood pressure and prevent heart failures. Inotropic agents to treat heart failures like dopamine, digitalis, dobutamine nitrates for anginal pains; mainly it is the nitroglycerin.

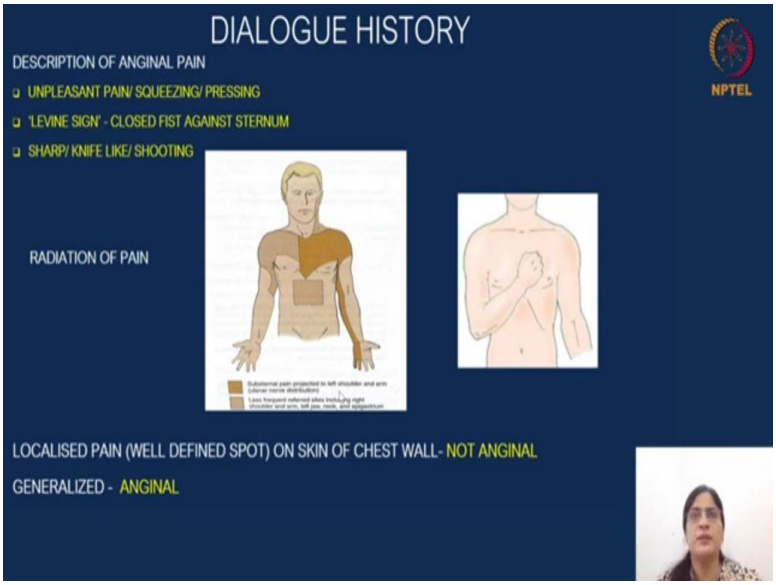
(Refer Slide Time: 29:17)





So, these are just the illustrations. This is the beta blocker propranolol, most commonly given. Nifidipine is the calcium channel blocker. Verapamil is another commonly given calcium channel blocker.

(Refer Slide Time: 29:32)



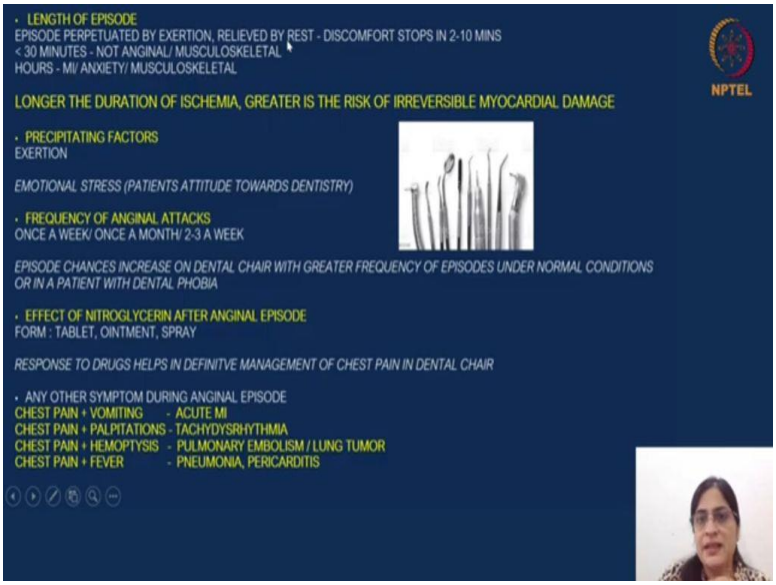

Now, after we are done with the medical history part we need to talk to the patient, if the patient is giving you the history of angina pectoris or myocardial infarction. What kind of pain what is the description of the patient pain, which is important for us to understand? So, the patient would describe an anginal pain maybe as an unpleasant pain, or squeezing pain, or a pressing kind of pain.

Levine sign is very important; it is a kind of closed fist against the sternum. The angina pectoris, or more commonly the myocardial infarction patients will give you a demonstration of this Levine sign; the pain can be sharp also, knife like also, a shooting also.

So, the pain radiates to the left shoulder and left arm along the course of the ulnar nerve. And less frequent sides would be right shoulder and arm, left jaw, neck, and epigastrium. The pain is localized. So, even if it is a sharp knife like pain, it will be a localized pain in case of non-angina.

In case it is anginal pain, if it is sharp, knife like or shooting; it will be generalized, not localized. So, localized pain which the patient is able to pinpoint to a well defined spot on the skin of the chest wall is not in anginal pain; the pain in angina is generalized. So, this is how you differentiate an anginal pain from a non-anginal pain; this we have already discussed in the beginning of the talk.

(Refer Slide Time: 31:05)




Coming to the length of the episode, this is important for us to know. So, we must ask the patient about the duration of the pain. So, episode is usually and we must ask the aggravating factors is perpetuated by exertion, and it is relieved by rest; and the discomfort usually stops in 2 to 10 minutes. So, the pain in case it is more than 15 minutes going up to 30 minutes is not an anginal pain; it could be musculoskeletal.

And if the pain is going up to hours, it can be myocardial infarction; it can be the patient when the patient is over anxious or a musculoskeletal pain. So, we must understand that the longer the duration of ischemia, greater is the risk of irreversible myocardial damage. So, basically angina pectoris untreated, unattended can lead to myocardial infarction.

So, here we have the precipitating factors like exertion, emotional stress; we must try to understand or gauge the patient's attitude towards dentistry. Whether the patient is very fearful or the patient is having some dental phobia and over anxious patients. We must ask the frequency of anginal attacks of the patient, whether the anginal attacks are happening once a week, once a month, or 2 to 3 times a week.

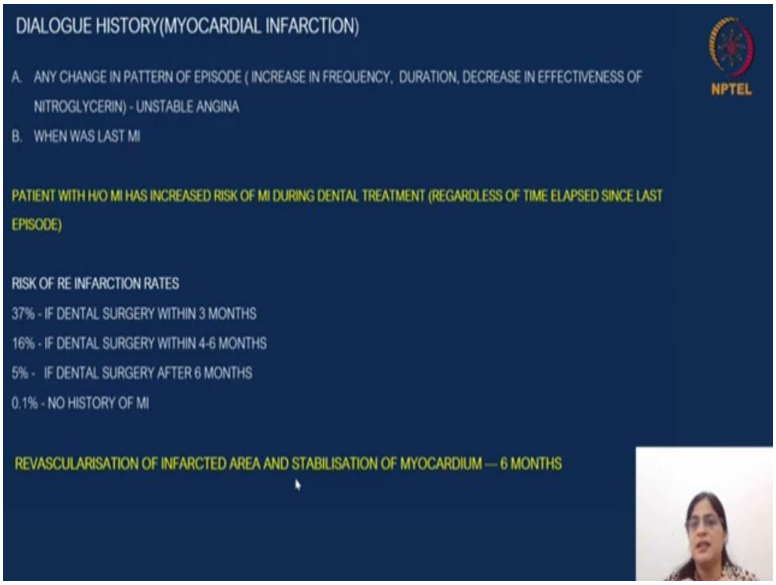
The chance of an acute angina episode increases on the dental chair with a greater frequency of episodes under normal conditions, or in a patient with dental phobia; that is why all this history is very relevant to us. What is the effect we must ask the patient? What is the effect of nitroglycerin after an anginal episode?

First of all, they must know when which form of nitroglycerin the patient is having whether it is a tablet or in spray, or topical ointment? So, this kind of whether it is a response to a tablet

or a spray? So, this will help in definitive management of chest pain in case it happens in the dental chair. We must ask the patients of any other symptoms associated during anginal episode that will hint us towards the other ailments.

Like if the chest pain is accompanied with vomiting, it can be an acute myocardial infarction. If the chest pain is with palpitations, it could be tachydysrhythmia. If the chest pain is with hemoptysis that means the cough with blood in sputum; it could be pulmonary embolism or a lung tumor also. And if there is a femur fever accompanied with chest pain, it could be pneumonia, it can be pericarditis. So, the associated symptoms also are very important when we are asking the patient about the history of chest pain.

(Refer Slide Time: 33:43)




So, what is different in myocardial infarction when we try to ask the patient, other than what we asked the patient for angina pectoris? We must add on to some more questions when we are dealing with a patient with the history of myocardial infarctions. And the added questions would be if the patient has noted any change in pattern of episode of pain. If the, now what can be the change in pattern? It could be either the increase in frequency it could be increase in duration, or there could be a decrease in effectiveness of like nitroglycerin.

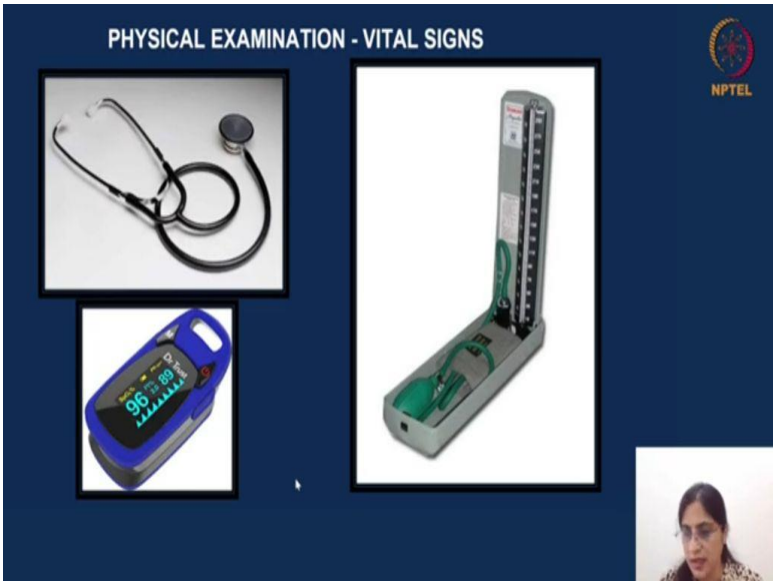
That means the patient is moving towards unstable angina; and unstable angina is a high risk for myocardial infarction. And we must ask the patient when about the history of last myocardial infarction; because that duration is very important for us. So, the patient with history of myocardial infarction has increased risk of myocardial infarction during the dental treatment; regardless of the time elapsed since last episode.

So, any history whether it is a year old history or a two year old history, that is also important for us to prevent any kind of attack during the dental treatment. And the risk of reinfarction rate is never zero. It is maximum, in case you try to do a dental procedure within the three months of myocardial infarction; while it is still 0.1 percent even if the patient gives no history of myocardial infarction.

So, there is always a risk of myocardial infarction in high risk patients to happen in the dental change. And why we always tell you that six months is the ideal time, you have to wait for any elective dental procedures to be done in case by the patient's gives you the history of myocardial infarction.

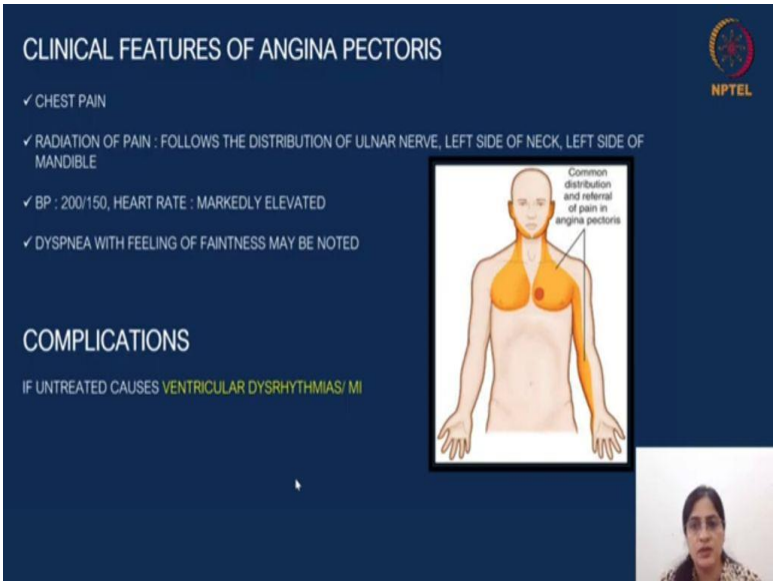
Because, the revascularization what we talked talk about the adequacy of the collateral circulation, it of the infected area happens in six months. The stabilization of myocardium happens in six months. That is why it is recommended that at least six months you have to wait for any elective dental procedure to be done in a patient with a history of myocardial infarction.

(Refer Slide Time: 35:58)




So, once we are done with the history of the pain and the related features, we come to the physical examinations. So, we need to record the blood pressure, pulse rate, respiratory rate, temperature, oxygen saturation for the patient before we start with the procedure. And it is a good practice, especially in cases of history of myocardial infarction that you check the vital signs, even after the procedure; and make the patient wait in the recovery room before you discharge the patient.

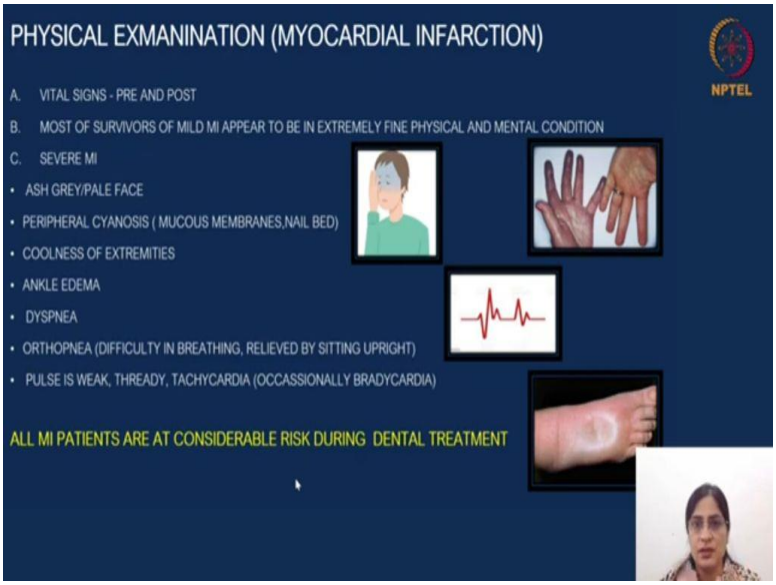
(Refer Slide Time: 36:31)




So, in the physical examination, after the vital signs, we must know what would be the clinical features of angina pectoris in case it happens in chair? So what is happening with the patient you should be able to recognize it is an anginal attack. First of all, is the description of the chest pain is highly helpful. So, you will be able to make out whether the chest pain is of cardiac origin or non-cardiac origin.

Check for the radiation of the pain, which will be along the distribution of the ulnar nerve, the left side of the neck, left side of the mandible. The BP would be shooting to 200 by 150 millimeters of mercury. There could be a tachycardia, the patient might be dyspnea with the feeling of faintness also; so, syncope also could be present. So, if it goes untreated, it can cause ventricular dysrhythmias or myocardial infarction.

(Refer Slide Time: 37:25)




So, now let us come to the physical examination in case of the patient. If the patient has an myocardial infarction attack, or you can call it as a heart attack in layman's term in dental chair. So, vital signs we have already discussed; we have to check pre and post. So, there could be a mild MI or it could be a severe MI. So, mild MI patients and the survivors of mild MI patients would be extremely okay in physical and mental conditions.

While, a patient with a severe and MI will show changes in the face like Ash gray face, pale face; then would be peripheral cyanosis. Peripheral cyanosis may mean you need to check the mucous membranes and the nail beds. There will be coolness of extremities, there will be ankle edema, which you can note.

There will be dyspnoea, orthopnea. Orthopnea means with difficulty in breathing due to posture and it is usually relieved by sitting upright. The pulse will be weak, thready, and tacky; and occasionally you will see bradycardia. But, all patients whether mild MI patients or severe MI patients; all patients are at considerable risk during the dental treatment.