

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 Angina 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)

POTENTIAL CAUSES OF CHEST PAIN

ANGINA PECTORIS (MOST COMMON) NON CARDIAC (COMMON) MYOCARDIAL INFARCTION (LESS COMMON)

NON CARDIAC CAUSES

PERICARDITIS

- Inflammation of pericardium
- Etiology-viral infection
- Pain is substernal, aggravated on breathing and swallowing
- Often presence of fever before onset of pain
- Relieved by bending from waist

MUSCULOSKELETAL

- Resulting from muscle strain after exercise
- Localised, does not radiate, worsens by breathing and movement
- Relieved by heating pad and analgesic

ESOPHAGITIS

- Substernal, epigastric burning pain
- Aggravated by eating or lying down after a meal
- Relieved by antacids

POTENTIAL CAUSES OF CHEST PAIN

ANGINA PECTORIS (MOST COMMON) NON CARDIAC (COMMON) MYOCARDIAL INFARCTION (LESS COMMON)

NON CARDIAC CAUSES

PULMONARY EMBOLISM

- Due to sudden occlusion of blood vessel within lungs by an embolus
- Sudden, severe chest pain
- Acute life threatening situation, Coughing of blood tinged sputum

DISSECTING AORTIC ANEURYSM

- Less common
- Sudden, severe chest pain at the onset, spreads up and down the chest and back over few hours
- May lead rapidly to death

PAIN OF ACUTE INDIGESTION AND "GAS"

- Primarily in upper epigastric region
- Gas pain- sharp, knife like, increases in intensity with breathing

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.

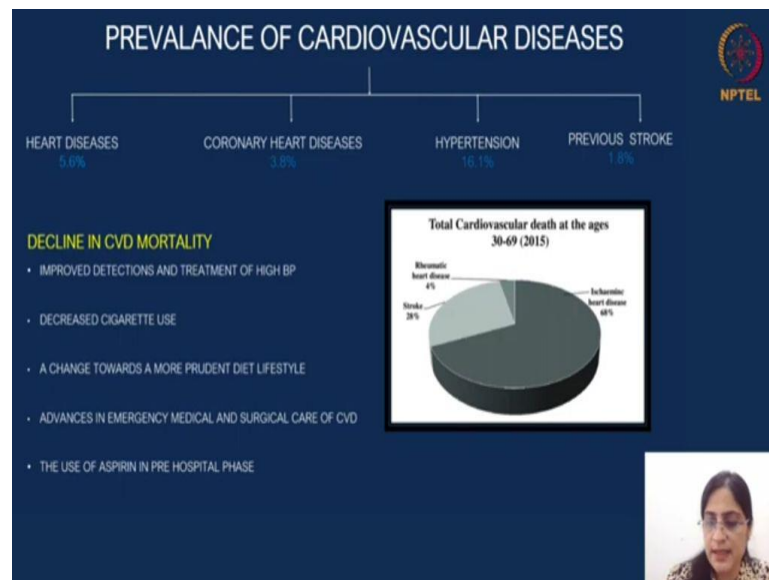
The etiology is viral infection, the location of pain is again substernal, aggravated on breathing and swallowing. There is often presence of fever before the onset of pain and it is relieved by bending from waste. Another common non cardiac cause is pain of acute indigestion and gas;

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 tear 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)



PATIENTS SUFFERING FROM CVD REPRESENT A GREAT POTENTIAL RISK DURING ROUTINE DENTAL TREATMENT - WHY ???

- MOST OF THEM ARE AMBULATORY
- SIGNIFICANT NUMBER IS ASYMPTOMATIC
- SOME ARE UNAWARE ABOUT THEIR CVD


SO, ANY DENTAL PROCEDURE WHICH CAN INCREASE WORKLOAD OF PATIENT'S CVS IS POTENTIALLY DANGEROUS!

The slide features two illustrations: one of a dentist in a white coat talking to a patient in a green suit, and another of an elderly couple looking confused with question marks around them. The NPTEL logo is in the top right corner.


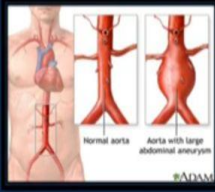

Why do the patients suffering from ischemic heart disease represent a great risk during routine dental treatment? Because, these patients would be ambulatory; that means walking around normally doing their routine daily procedures. These patients would be asymptomatic and some of them would not know 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)

PREDISPOSING FACTORS OF CARDIO - VASCULAR DISEASES



- **ATHEROSCLEROSIS**
- **PERIPHERAL VASCULAR DISEASES**
- **ANEURYSM OF ABDOMINAL AORTA**



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)

ATHEROSCLEROSIS

DEFINITION : THICKENING AND HARDENING OF MEDIUM AND LARGE SIZED ARTERIES , CONSTITUTES A LARGE PROPORTION OF CAUSES OF ISHAEMIC HEART DISEASES

RISK FACTORS FOR ATHEROSCLEROTIC DISEASE

1. DYSLIPIDEMIA
2. SMOKING
3. HIV
4. DIABETES
5. OBESITY AND LACK OF EXERCISE
6. MENTAL STRESS
7. ESTROGEN STATUS



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)

The slide is titled "DYSLIPIDEMIA" and "SMOKING". It features a dark blue background with white and yellow text. In the top right corner, there is a small circular logo with the text "NPTEL" below it. The slide contains the following text:

- TOTAL CHOLESTROL : > 200-239 (BORDERLINE HIGH)
> 240 (HIGH, TWICE THE RISK OF CHD)
- HDL : < 40 (MAJOR RISK FACTOR FOR CHD)
- LDL : > 130 mg/dl (BORDERLINE HIGH)
> 190 mg/dl (VERY HIGH)
- TRIGLYCERIDES : 150-199 mg/dl (BORDERLINE HIGH)
>500 mg/dl (VERY HIGH)

In the center, there are two circular icons: one showing a cross-section of an artery with a plaque, and another showing a lit cigarette. Below the icons, the word "SMOKING" is written in large white letters.

- TOBACCO SMOKING IS A MAJOR RISK FACTOR FOR ACUTE MI
- DIRECT RELATIONSHIP BETWEEN CAD INCIDENCES AND NUMBER OF CIGARETTES SMOKED DAILY
- RISK OF FIRST MI (HEART ATTACK) IS REDUCED BY NEARLY 65% WITH SMOKING CESSATION

In the bottom right corner, there is a small video inset showing a woman speaking.

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)

The slide is dark blue with white and yellow text. At the top, it says 'HYPERTENSION'. Below that, there are three bullet points in yellow: 'META-ANALYSIS SHOWS 29% INCREASE IN RISK OF CHD FOR EVERY 7mmHg ELEVATION OF DIASTOLIC BP', 'PHARMACOLOGICAL REDUCTION OF DIASTOLIC BP BY 5-6mmHg APPEAR TO REDUCE RISK OF CHD BY 14%', and 'DAMAGE TO THE ARTERIES OVER THE YEARS CANNOT BE UNDONE, BUT ATHEROSCLEROTIC PROCESS CAN BE SLOWED DOWN'. To the right of the first two points is a red heart icon with a white pulse line. In the top right corner is a circular logo with 'NPTEL' written below it. Below the hypertension section, it says 'INSULIN RESISTANCE AND DIABETES'. Below that, there are two bullet points in yellow: 'DIABETICS HAVE 3 TO 5 FOLD INCREASED RATE OF FUTURE CARDIO VASCULAR EVENTS DUE TO ATHEROSCLEROSIS IN MAJOR ARTERIES' and 'INSULIN RESISTANCE PROMOTES ATHEROSCLEROSIS'. To the right of the first point is an icon of a hand holding a blue pill. In the bottom right corner, there is a small video inset showing a woman with glasses and a patterned top.

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)

The slide is titled "EXERCISE AND OBESITY" and "MENTAL STATUS". It contains the following text:

- REGULAR PHYSICAL EXERCISE DECREASES MYOCARDIAL O₂ DEMAND, THE EXERCISE CAPACITY DECREASES CORONARY RISK
- MECHANISMS : FAVORABLE EFFECTS ON BP, WEIGHT CONTROL, LIPID PROFILES, IMPROVED GLUCOSE TOLERANCE

MENTAL STATUS

- ADRENERGIC STIMULATION INCREASES MYOCARDIAL O₂ DEMAND → AGGRAVATES MYOCARDIAL ISCHEMIA
- CAUSES CORONARY VASOCONSTRICTION IN ATHEROSCLEROTIC CORONARY ARTERIES
- ALTERATIONS IN THROMBOSIS AND COAGULATION → FAVORS CLOT FORMATION

DENTAL CLINIC ENVIRONMENT CAN BE STRESSFUL FOR FEARFUL PATIENTS

The slide also features an illustration of people exercising, the NPTEL logo, and a small video inset of a woman speaking.

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.



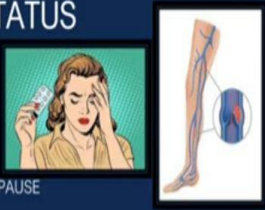
Coming to the mental status of the patient. When we talk about the mental status, we are talking about the patients who are over anxious, or they have a phobia towards dental treatment. So, what happens in such patients if the patients are very anxious? They will have release of adrenaline, which is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

ESTROGEN STATUS

- DELAYS THE PROGRESSION OF ATHEROSCLEROSIS
- CAD INCREASES IN POST MENOPAUSE THAN PRE MENOPAUSE
- ORAL CONTRACEPTIVES AMONGST YOUNG WOMEN INCREASES RATE OF DEEP VEIN THROMBOSIS, MI, STROKE
- ESTROGEN USE AS HORMONAL REPLACEMENT THERAPY IN POST MENOPAUSAL WOMEN DECREASES CARDIOVASCULAR RISK BY 35-45%



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)

ANGINA PECTORIS

- ANGINA (LATIN WORD) - SPASMODIC, CRAMP LIKE OR CHOKING FEELING/ SUFFOCATING PAIN
- PECTORIS (LATIN WORD) - CHEST
- FIRST EVER USED BY DR. WILLIAM HEBERDEN (1768) TO DISTINGUISH STRANGLING FEELING OF ANGINA FROM "DOLOR" WHICH MEANS PAIN.

HOW DO YOU DEFINE ANGINA PECTORIS ?

A CHARACTERISTIC THORACIC PAIN, USUALLY SUBSTERNAL, PRECIPITATED BY EXERCISE, EMOTION OR A HEAVY MEAL, RELIEVED BY VASODILATOR DRUGS AND A FEW MINUTES REST AND A RESULT OF A MODERATE INADEQUACY OF CORONARY CIRCULATION

- PATIENTS COMMONLY DESCRIBE THIS PAIN AS CRUSHING, HEAVY, SUFFOCATING, CONSTRICTING, SQUEEZING AND DULL OR ACHING DISCOMFORT



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)

IT IS IMPORTANT TO DISTINGUISH ANGINAL CHEST PAIN FROM CHEST PAIN DUE TO OTHER REASONS LIKE INTERCOSTAL MUSCLE SPASMS



CHEST PAIN WHICH IS SHARP, KNIFE LIKE LASTING FOR HOURS WITH BREATHING AND IS LOCALISED CANNOT BE PAIN OF ISCHEMIC ORIGIN

CLINICAL CHARACTERISTICS OF ANGINA

CHARACTERISTIC	MORE LIKELY TO BE ANGINA	LESS LIKELY TO BE ANGINA
TYPE OF PAIN	DULL PRESSURE	SHARP STABBING
DURATION	2-5 min, ALWAYS < 15-20 min	SECOND OR HOURS
ONSET	GRADUAL	RAPID
LOCATION	SUBSTERNAL	LATERAL CHEST WALL BACK
REPRODUCIBLE	WITH EXERTION	WITH INSPIRATION
ASSOCIATED SYMPTOMS	PRESENT	ABSENT
PALPATION OF CHEST WALL	NOT PAINFUL	PAINFUL




So, we need to differentiate an 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)

PRECIPITATING FACTORS IN ANGINA PECTORIS

ACUTE ANGINAL EPISODES CAN BE PRECIPITATED BY CERTAIN FACTORS WHICH CAN INCREASE MYOCARDIAL O₂ DEMAND WHICH CORONARY ARTERIES ARE UNABLE TO SUPPLY

TRIGGERS 4E'S

1. EMOTION(ANXIETY/EXCITEMENT)
2. EXERCISE(PHYSICAL ACTIVITY)
3. EXPOSURE TO COLD/HOT&HUMID
4. EATING(LARGE MEALS)



OTHERS: CAFFEINE INGESTION/FEVER/ANEMIA/THYROTOXICOSIS/CIGARETTE SMOKING/SMOG/HIGH ALTITUDES

RELIEVED BY REST OR ADMINISTRATION OF NITROGLYCERIN




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)

TYPES OF ANGINA PECTORIS

- **STABLE ANGINA** IS MOST COMMON. USUALLY CAUSED BY OBSTRUCTION OF CORONARY ARTERIES BY ATHEROMATOUS PLAQUE.
- **VARIANT ANGINA** USUALLY OCCURS WHEN PATIENT IS AT REST, OFTEN ASSOCIATED WITH DYSRHYTHMIAS. MOST COMMON IN FEMALES <50 YEARS, WHEREAS STABLE ANGINA IS UNCOMMON IN THIS AGE GRP. SIGNS AND SYMPTOMS CAN BE SYNCOPE, DYSPNOEA, PALPITATIONS.
- **UNSTABLE ANGINA** IS INTERMEDIATE BETWEEN STABLE ANGINA AND ACUTE MYOCARDIAL INFARCTION. IT IS AGAIN DUE TO PROGRESSION OF ATHEROSCLEROSIS. *IT IS CALLED UNSTABLE WHEN ANGINA PECTORIS OCCURS AT REST, PAIN LASTING UPTO 30 MINUTES IF NOT INTERRUPTED BY NITROGLYCERIN OR IT CAN BE A FRANK PAIN OF NEW ONSET OR A MORE SEVERE PROLONGED AND FREQUENT THAN PREVIOUSLY.*

ANGINAL SYNDROME	SYNONYMS	PRECIPITATING FACTORS	DURATION	RESPONSE TO NITROGLYCERINE
STABLE	CHRONIC/CLASSIC /EXERTIONAL	EMOTIONAL STRESS,PHYSICAL EXERTION,COLD WEATHER	1-15 MINUTES	GOOD
VARIANT	PRINZMETAL'S/ ATYPICAL/ VASOSPASTIC	CORONARY ARTERY SPASM	VARIABLE	GOOD
UNSTABLE	PREINFARCTION/ CRESCENDO	ANY FACTOR OR NO FACTOR	UP TO 30 MINUTES	QUESTIONABLE


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)




ANGINA IS CLINICALLY IMPORTANT TO DENTISTRY AS IT IS A SIGN OF SIGNIFICANT DEGREE OF CAD

WHY PATIENT WITH A HISTORY OF ANGINA IS AT INCREASED RISK DURING DENTAL TREATMENT?

BECAUSE ANY FACTOR WHICH INCREASES MYOCARDIAL REQUIREMENTS CAN PRECIPITATE AN ACUTE EPISODE OF CHEST PAIN WHICH CAN ULTIMATELY LEAD TO MI/ DYSRHYTHMIA/ CARDIAC ARREST

EMOTIONAL AND PHYSICAL STRESS ARE MAJOR FACTORS WHICH CAN INCREASE CARDIAC WORKLOAD CAUSING CHEST PAIN DURING DENTAL TREATMENT, SO ELIMINATION OF THE STRESS IS THE PREVENTIVE MEASURE

- IN ORDER TO TREAT A PATIENT WITH CHEST PAIN FOR ANGINA, THEY MUST HAVE A PRIOR HISTORY OF ANGINA PECTORIS.
- ALL INSTANCES OF FIRST TIME CHEST PAIN OCCURRING IN A DENTAL SETTING REQUIRE IMMEDIATE ACTIVATION OF EMERGENCY MEDICAL SERVICES(EMS).

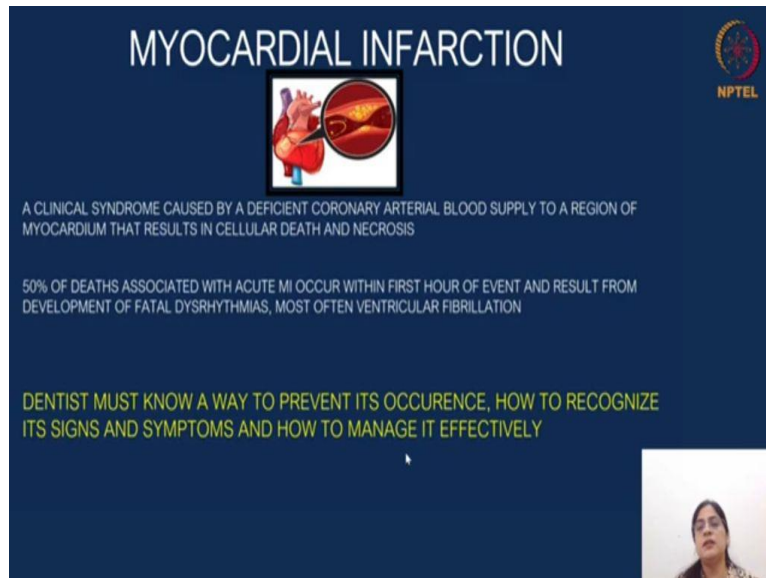


Why a patient with a history of angina is important to us is clinically relevant to us; and why he or she is at increased risk during dental treatment? Because any factor, basically here the factors when we are doing a routine dental procedure or any dental procedure, we are talking about the factors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



MYOCARDIAL INFARCTION

A CLINICAL SYNDROME CAUSED BY A DEFICIENT CORONARY ARTERIAL BLOOD SUPPLY TO A REGION OF MYOCARDIUM THAT RESULTS IN CELLULAR DEATH AND NECROSIS

50% OF DEATHS ASSOCIATED WITH ACUTE MI OCCUR WITHIN FIRST HOUR OF EVENT AND RESULT FROM DEVELOPMENT OF FATAL DYSRHYTHMIAS, MOST OFTEN VENTRICULAR FIBRILLATION

DENTIST MUST KNOW A WAY TO PREVENT ITS OCCURENCE, HOW TO RECOGNIZE ITS SIGNS AND SYMPTOMS AND HOW TO MANAGE IT EFFECTIVELY

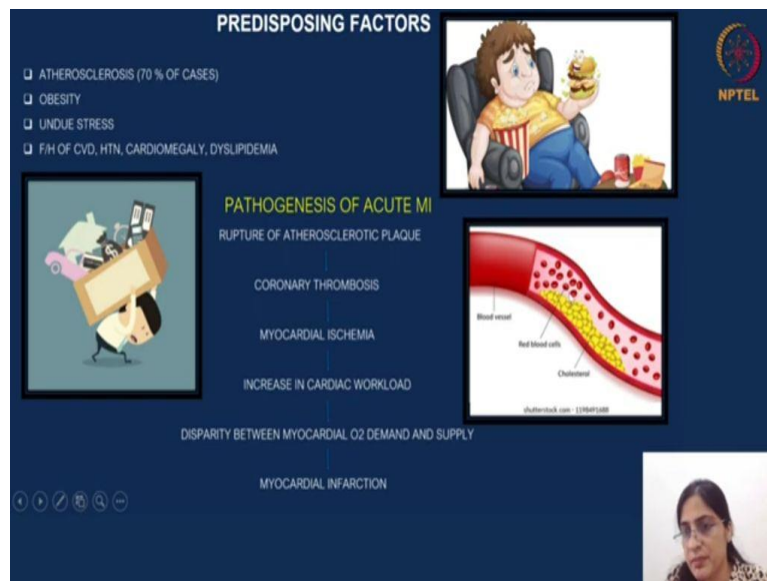
NPTEL

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)

The slide features a dark blue background with white and yellow text. At the top, the title 'LOCATION AND EXTENT OF INFARCTION' is centered. Below it, a vertical flowchart lists the following points: 'MOST COMMON SITE OF THROMBOSIS', 'ANTERIOR DESCENDING BRANCH OF LEFT CORONARY ARTERY', 'OCCLUSION OF VESSEL', 'INFARCTION (ABSENCE OF ADEQUATE COLLATERAL CIRCULATION)', and 'MYOCARDIAL NECROSIS THROUGHOUT DISTRIBUTION OF OCCLUDED ARTERY'. To the right of this text is an anatomical diagram of the heart showing the coronary arteries. Labels include: 'Right coronary artery (RCA) - supplies the right ventricle and the posterior part of the septum', 'Left anterior descending artery (LAD) - supplies the anterior wall of the left ventricle and the anterior part of the septum', 'Left circumflex artery (LCx) - supplies the lateral wall of the left ventricle', and 'Left posterior descending artery (LPD) - supplies the posterior wall of the left ventricle and the posterior part of the septum'. The NPTEL logo is in the top right corner. A small video inset in the bottom right shows a woman speaking.

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.

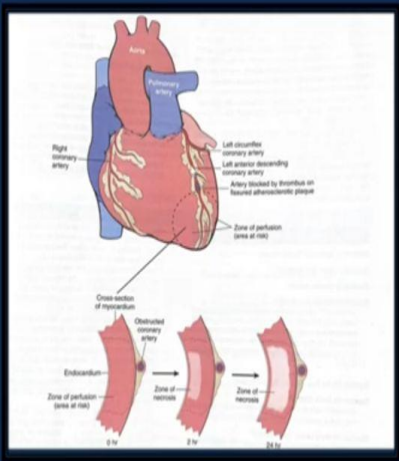
(Refer Slide Time: 19:45)

FACTORS DETERMINING EXTENT OF INFARCTION

- ANATOMIC DISTRIBUTION OF OCCLUDED VESSELS
- ADEQUACY OF COLLATERAL CIRCULATION
- ONE VESSEL V/S MULTIVESSEL INVOLVEMENT
- PREVIOUS INFARCTION IF ANY



NPTEL




Right coronary artery
Left circumflex coronary artery
Left anterior descending coronary artery
Artery blocked by thrombus in tunneled atherosclerotic plaque
Zone of perfusion (area at risk)

Cross section of myocardium
Endocardium
Zone of perfusion (area at risk)
Zone of jeopardy
Zone of infarction

0 hr
8 hr
24 hr

NPTEL

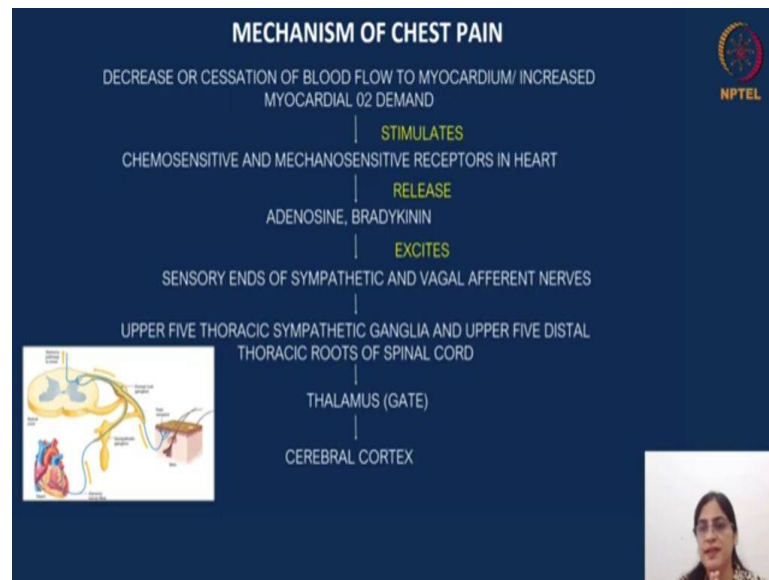


So, what determines the extent of infarction or the area of the myocardial damage, the distribution of occluded vessels? What is the adequacy of collateral circulation at that time? Whether it is one vessel involved or multiple vessels involved and if there was any preexisting infarction?

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)

WHAT HAVE WE LEARNED

- ✓ DIFFERENTIATE CARDIAC PAIN (IHD/CHD) FROM NON CARDIAC PAIN
- ✓ MECHANISM OF CHEST PAIN
- ✓ RISK FACTORS ASSOCIATED WITH ANGINA PECTORIS & MI
- ✓ DIFFERENCE BETWEEN ANGINA PECTORIS & MI
- ✓ PATHOPHYSIOLOGY OF ANGINA PECTORIS & MI
- ✓ ANGINA PECTORIS & MI – POTENTIAL RISK DURING DENTAL PROCEDURE

NPTEL

Navigation icons: back, forward, search, refresh, home, close.



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 infarction is a potential risk during dental procedure?

(Refer Slide Time: 22:31)

TWO SCENARIOS




TWO SCENARIOS

PREVENTION MANAGEMENT

PREVENTION

MEDICAL HISTORY, DIALOGUE HISTORY AND PHYSICAL EXAMINATION IS IMPORTANT AT FIRST DENTAL VISIT

NPTEL



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)

MEDICAL HISTORY

A. DISEASES

1. CHEST PAIN OR SHORTNESS OF BREATH , SWOLLEN ANKLES
2. HEART DISEASE
3. HEART ATTACK/ STROKE/ HIGH BLOOD PRESSURE
4. FAMILY HISTORY OF DIABETES, HEART DISEASES, TUMORS
5. THYROID/ ADRENAL/ DIABETES

FREQUENTLY ASSOCIATED WITH ATHEROSCLEROSIS/ANGINA

B. HOSPITALISATION/ SURGERIES - CAUSE AND TREATMENT

PATIENTS WITH PERSISTENT SYMPTOMS DESPITE ADEQUATE MEDICAL THERAPY ARE CONSIDERED FOR CORONARY REVASCULARISATION (PTCA, CABG)

NPTEL

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)

c. DRUGS

PATIENTS WITH ANGINA PECTORIS ARE ON VARIOUS DRUGS TO DECREASE THE FREQUENCY OF ACUTE EPISODES OR TERMINATE THE ACUTE EPISODE

COMMONLY PRESCRIBED DRUGS ARE ANTIPLATELET AGENTS

ASPIRIN (80-325 MG DAILY) → LESS CARDIOVASCULAR MORTALITY BY 30%-50%

ASPIRIN + CLOPIDOGREL (75 MG) → 16% MORE REDUCTION IN CV EVENTS.

NITROGLYCERINE – ACUTE ANGINAL EPISODES

CALCIUM CHANNEL BLOCKERS ARE INDICATED IN CASE OF ANY ADVERSE OR INADEQUATE RESPONSE / CI TO BETA BLOCKERS OR IN PRINZMETAL'S ANGINA.

Clopidogrel & Aspirin Tablets
Clopidogrel A-75

Nitroglycerin sublingual spray
USP 0.5 mg
Mylan

NPTEL

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)



ASPIRIN/CLOPIDOGREL

DOSAGE

- Acute Ischaemic Stroke: 150-300mg as a single dose given within 48hrs of onset and 75-150mg daily reduces the risk of having another stroke.
- Myocardial infarction: 150-300mg for initial management. While a dose of 75-150mg is given for long-term management to reduce the rate of reinfarction.

SOLD AS ECOSPRIN, PLAVIX, CLOPIVAS, CLOPIVAS AP

The slide features a dark blue background with a white text box for dosage information. It includes images of a spilled pill bottle, a box of Aspirin Clopidogrel Tablets, and several boxes of Plavix. The NPTEL logo is in the top right corner, and a small video inset of a woman is in the bottom right.



NEWER DRUGS FOR MANAGEMENT OF ANGINA AND MI

ANTIPLATELET THERAPY

- PRASUGREL
- TICAGRELOR
- ABCIXIMAB
- EPTIFIBATIDE
- TIROFIBAN

ANTICOAGULANT THERAPY

- ENOXAPARIN
- BIVALIRUDIN

The slide features a dark blue background with text and images of drug packaging. It includes images of Prasugrel tablets, Ticagrelor tablets, and Enoxaparin Sodium Injection. The NPTEL logo is in the top right corner, and a small video inset of a woman is in the bottom right.



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)

BETA BLOCKERS AND CALCIUM CHANNEL BLOCKERS TO PREVENT OR TREAT ANGINA PECTORIS

GENERIC NAME	PROPRIETARY NAME	SIDE EFFECTS
BETA BLOCKERS (CARDIO-SELECTIVE)		FOR ALL BETA BLOCKERS-SEVERE BRADYCARDIA, AV CONDUCTION DEFECTS, LEFT VENTRICULAR FAILURE
ATENOLOL	TENORMIN	
METOPROLOL CARVEDILOL	LOPRESSOR COREG	
BETA BLOCKER (NON SELECTIVE)		FOR NON SELECTIVE BETA BLOCKERS-BRONCHOSPASM
NADOLOL PROPRANOLOL	CORGARD INDERAL	
CALCIUM CHANNEL BLOCKERS		PERIPHERAL EDEMA, HYPOTENSION, HEADACHE, DIZZINESS
NIFEDIPINE	ADALAT	
AMLODIPINE	NORVASC	
NICARDIPINE	CARDENE	
NITRENDIPINE	BAYPRESS	
VERAPAMIL	VERELAN	
DILTIAZEM	CARDIZEM	



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)

MEDICATIONS USED FOR PATIENTS WHO HAVE HAD MYOCARDIAL INFARCTION

- ANGIOTENSIN CONVERTING ENZYME INHIBITORS : REDUCE BLOOD PRESSURE AND CARDIAC AFTERLOAD RESULTING IN A REDUCTION OF MYOCARDIAL OXYGEN DEMAND, GIVEN FOR ACUTE MI FOR 8 WEEKS AND PERMANENTLY FOR REDUCED LV FUNCTION
- LIPID LOWERING AGENT : STATINS
- ANTICOAGULANTS : WARFARIN DURING FIRST 3 MONTHS AFTER LARGE MFS
- DIURETICS : TO MANAGE HF & HIGH BP

DRUG CATEGORY	EXAMPLES	RATIONALE
BETA BLOCKERS	PROPRANOLOL, METOPROLOL	DECREASES LIKELINESS OF SUDDEN DEATH
CALCIUM-CHANNEL BLOCKERS	DILTIAZEM, VERAPAMIL	PREVENT REINFARCTION AND REDUCE MORTALITY RATE
ANTIPLATELET AGENTS	ASPIRIN, CLOPIDOGREL	REDUCES INCIDENCE OF SUDDEN DEATH
ANTICOAGULANTS	WARFARIN	REDUCES INCIDENCE OF ARTERIAL EMBOLI
ACE INHIBITORS	CAPTOPRIL	PREVENT LEFT VENTRICULAR DILATION
DIURETICS	HYDROCHLOROTHIAZIDE	HIGH BLOOD PRESSURE, HEART FAILURE
INOTROPIC DRUGS	DIGITALIS, DOPAMINE, DOBUTAMINE	HEART FAILURE
NITRATES	NITROGLYCERINE	ANGINAL PAINS

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)




PROPRANOLOL

DOSAGE OF PROPRANOLOL


- Starting dose: 40-80 mg once daily
- Max. dose/day: 240 mg
- If satisfactory response is not obtained within 4-6 weeks, after reaching the maximal dose, therapy should be discontinued
- Taper slowly to avoid rebound headache and adrenergic side effects
- Max. duration: 9 to 12 months






The slide also includes an image of a box of Propranolol Tablets LP and a small video inset of a presenter in the bottom right corner.


NIFEDIPINE



□ Dose of nifedipine: an initial oral dose of 20 mg followed by 10–20 mg three to four times daily, adjusted according to uterine activity for up to 48 hours. A total dose above 60 mg appears to be associated with a three- to four-fold increase in adverse events.





VERAPAMIL





ANTI-ARRHYTHMIC DRUGS



CLASS IV CALCIUM CHANNEL BLOCKERS



VERAPAMIL

- Oral administration – 20% bioavailability
- t_{1/2} 4-7 hrs
- Linear metabolism
- Dosage:
 - IV: 5-10 mg every 4-6 hrs or infusion of 0.4 ug/kg/min
 - Oral: 120-480 mg daily, divided in 3-4 doses
- Toxicity: AV block, atrial fib, ventricular arrhythmias
- Toxicity: AV block, can get sinus arrest, constipation, laxative, nervousness, peripheral edema





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

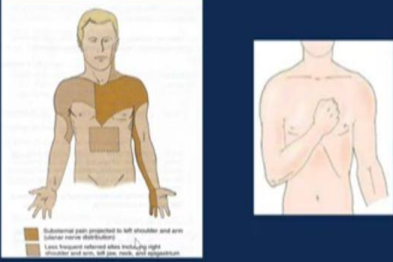
(Refer Slide Time: 29:32)

DIALOGUE HISTORY

DESCRIPTION OF ANGINAL PAIN


- UNPLEASANT PAIN/ SQUEEZING/ PRESSING
- 'LEVINE SIGN' - CLOSED FIST AGAINST STERNUM
- SHARP/ KNIFE LIKE/ SHOOTING


RADIATION OF PAIN



LOCALISED PAIN (WELL DEFINED SPOT) ON SKIN OF CHEST WALL - **NOT ANGINAL**

GENERALIZED - **ANGINAL**





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)

• LENGTH OF EPISODE
EPISODE PERPETUATED BY EXERTION, RELIEVED BY REST - DISCOMFORT STOPS IN 2-10 MINS
< 30 MINUTES - NOT ANGINAL/ MUSCULOSKELETAL
HOURS - MI/ ANXIETY/ MUSCULOSKELETAL

LONGER THE DURATION OF ISCHEMIA, GREATER IS THE RISK OF IRREVERSIBLE MYOCARDIAL DAMAGE

• PRECIPITATING FACTORS
EXERTION
EMOTIONAL STRESS (PATIENTS ATTITUDE TOWARDS DENTISTRY)

• FREQUENCY OF ANGINAL ATTACKS
ONCE A WEEK/ ONCE A MONTH/ 2-3 A WEEK

EPISODE CHANGES INCREASE ON DENTAL CHAIR WITH GREATER FREQUENCY OF EPISODES UNDER NORMAL CONDITIONS OR IN A PATIENT WITH DENTAL PHOBIA

• EFFECT OF NITROGLYCERIN AFTER ANGINAL EPISODE
FORM : TABLET, OINTMENT, SPRAY

RESPONSE TO DRUGS HELPS IN DEFINITIVE MANAGEMENT OF CHEST PAIN IN DENTAL CHAIR

• ANY OTHER SYMPTOM DURING ANGINAL EPISODE
CHEST PAIN + VOMITING - ACUTE MI
CHEST PAIN + PALPITATIONS - TACHYDYSRHYTHMIA
CHEST PAIN + HEMOPTYSIS - PULMONARY EMBOLISM / LUNG TUMOR
CHEST PAIN + FEVER - PNEUMONIA, PERICARDITIS

NPTEL

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 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)

DIALOGUE HISTORY(MYOCARDIAL INFARCTION)

A. ANY CHANGE IN PATTERN OF EPISODE (INCREASE IN FREQUENCY, DURATION, DECREASE IN EFFECTIVENESS OF NITROGLYCERIN) - UNSTABLE ANGINA

B. WHEN WAS LAST MI

PATIENT WITH H/O MI HAS INCREASED RISK OF MI DURING DENTAL TREATMENT (REGARDLESS OF TIME ELAPSED SINCE LAST EPISODE)

RISK OF RE INFARCTION RATES

- 37% - IF DENTAL SURGERY WITHIN 3 MONTHS
- 16% - IF DENTAL SURGERY WITHIN 4-6 MONTHS
- 5% - IF DENTAL SURGERY AFTER 6 MONTHS
- 0.1% - NO HISTORY OF MI

REVASCULARISATION OF INFARCTED AREA AND STABILISATION OF MYOCARDIUM — 6 MONTHS

NPTEL

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.

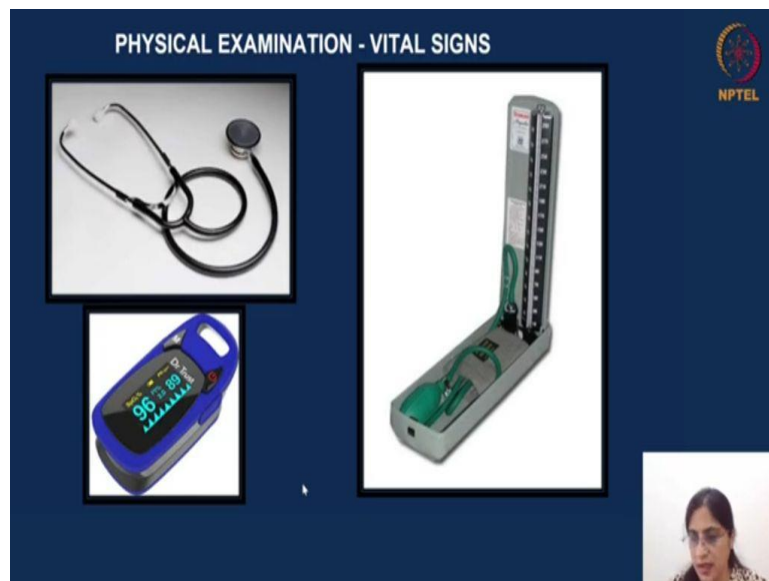
That means the patient is moving towards unstable angina; and unstable angina is a high risk for myocardial infarction. And we must ask the patient when about the history of last myocardial infarction; because that duration is very important for us. So, the patient with history of myocardial infarction 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.

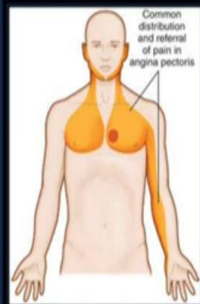
(Refer Slide Time: 36:31)

CLINICAL FEATURES OF ANGINA PECTORIS



- ✓ CHEST PAIN
- ✓ RADIATION OF PAIN : FOLLOWS THE DISTRIBUTION OF ULNAR NERVE, LEFT SIDE OF NECK, LEFT SIDE OF MANDIBLE
- ✓ BP : 200/150, HEART RATE : MARKEDLY ELEVATED
- ✓ DYSPNEA WITH FEELING OF FAINTNESS MAY BE NOTED

COMPLICATIONS

IF UNTREATED CAUSES VENTRICULAR DYSRHYTHMIAS/ MI



Common distribution and referral of pain in angina pectoris



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)

PHYSICAL EXAMINATION (MYOCARDIAL INFARCTION)







A. VITAL SIGNS - PRE AND POST

B. MOST OF SURVIVORS OF MILD MI APPEAR TO BE IN EXTREMELY FINE PHYSICAL AND MENTAL CONDITION

C. SEVERE MI

- ASH GREY/PALE FACE
- PERIPHERAL CYANOSIS (MUCOUS MEMBRANES, NAIL BED)
- COOLNESS OF EXTREMITIES
- ANKLE EDEMA
- DYSPNEA
- ORTHOPNEA (DIFFICULTY IN BREATHING, RELIEVED BY SITTING UPRIGHT)
- PULSE IS WEAK, THREADY, TACHYCARDIA (OCCASSIONALLY BRADYCARDIA)

ALL MI PATIENTS ARE AT CONSIDERABLE RISK DURING DENTAL TREATMENT



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.