RESEARCH ARTICLE

A PROSPECTIVE OBSERVATIONAL STUDY ON THE EVALUATION OF THERAPEUTIC STRATEGIES IN THE MANAGEMENT OF ATHEROSCLEROSIS: A COMPHREHENSIVE CLINICAL AND PHARMACOLOGICAL APPROACH

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Manuscript Info	Abstract
	Background: The goal of our study was to evaluate the comprehensive analysis of prospective study of the therapeutic management of Atherosclerosis. Arteriosclerosis is a type of vascular disease where the blood vessels carrying oxygen away from the heart (arteries) become damaged from factors such as hyperlipidemia, hypertension, diabetes and certain genetic influences. **Objective:** The objective is to evaluate various therapeutic strategies in the management of atherosclerosis focusing on clinical and pharmacological roles of antihyperlipidimic, antiplatelet, anticoagulant, antianginal, antidiabetic, antihypertensive and diuretics in the disease progression and patient outcome. **Methodology:** It was hospital based comprehensive study conducted by random selection of patients. A prospective observational study involving analysis of inpatients of cardiology department in multispeaciality hospital. All eligible patients diagnosed with atherosclerosis based on clinical evaluation and confirmed through angiographic reports were enrolled after obtaining informed consent. Baseline data including demographic details, medical history, lifestyle factors, and current medications were recorded.Angiogram reports were thoroughly assessed to evaluate the location, severity, and extent of arterial stenosis. Patients were then managed according to standard therapeutic strategies, including pharmacological treatments such as statins, antiplatelets, antihypertensives, and antidiabetic agents, along with non pharmacological approaches like lifestyle modifications.

RESULTS: A total of 100 cases of atherosclerosis were observed. Majority of cases were males (72%) compare to females (28%). The most often prescribed antihyperlipidimic is Atorvastatin (92%) compare to rosuvastatin (8%). The most frequently prescribed antiplatelet is Aspirin (52%) compare to clopidogril (24%). Most commonly prescribed anticoagulant is heparin

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(28%) compare to enoxaparin (4%). The most common social history is of Smokers (21%) , followed by Tobacco chewers(7%) , alcoholism (5%) , followed by no social history (67%). The most common eliological diagnons is of CAD TVD (77%) followed by CAD - DVD (26%) , followed by CAD - SVD (13%) , followed by ACS (8%) , followed by MI (7%) , followed by PAD (5%) .

CONCLUSION: It is clearly seen from results that Atorvastatin + Aspirin + Nicorandil is the most prominent drug combination. These were also drug of choice at the place of study given majority of patients during course of our study other classes of drugs such as Rosuvastatin and tirofiban which were rarely used.

Introduction:-
Atherosclerosis:
There is a medical condition known as atherosclerosis that has persisted for a long time and is characterized by inflammation in the arterial walls, lipid accumulation, inflammatory cells, and fibrous tissues. Therefore, plaque develops, which leads to cardiovascular disorders and obstruction of blood flow.[1] As plaques or atheromas accumulate within arteries, they gradually narrow and become less elastic. Plaques are made of fibrous material containing lipids, cholesterol, calcium, and cellular waste. These include the coronary, carotid, and peripheral arteries, which are considered targeted arteries.[2] In terms of morbidity and mortality, atherosclerosis is a major cause of heart attacks and strokes as well as of many other cardiovascular conditions. Due to its chronic nature and need for lifelong care, it causes over 17.9 million deaths annually, which has a significant impact on healthcare expenses.
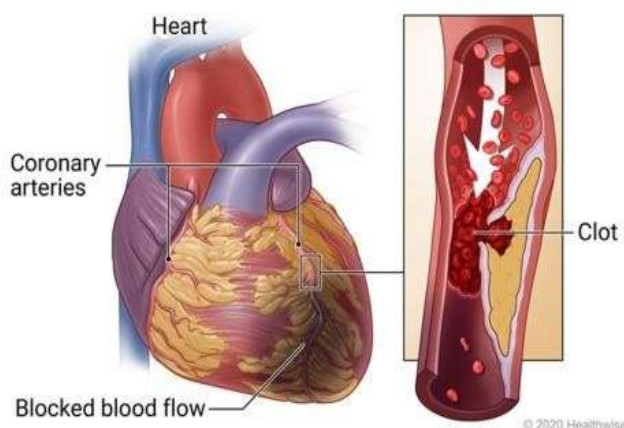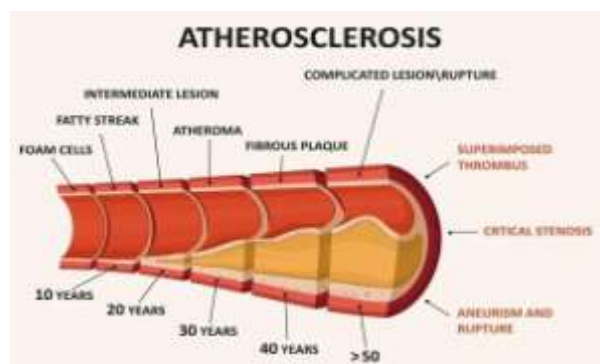
ETIOLOGY:-
Hypertension:
Atherosclerosis is a long-term, degenerative condition that causes plaque to build up inside artery walls, which can result in cardiovascular problem. Numerous associated risks are linked to its etiology.

Diabetes Mellitus:
Diabetes damages the activity of endothelial cells and raises oxidative stress by causing insulin resistance and elevated blood glucose. These elements play a part in plaque development and artery irritation.

High Cholesterol:
 High levels of LDL cholesterol are a substantial risk factor. Inflammation and the formation of foam cells inside arterial walls due to oxidized LDL cholesterol.

Smoking:
Smoking tobacco includes toxic chemicals that raise irritation, lower nitric oxide levels, and oxidatively destroy endothelium cells all of which may result in the formation of atherosclerosis.

Oxidative Stress:
Research has demonstrated that a larger ratio between ROS and antioxidants might exacerbate the destruction of endothelial cells. Oxidative stress and irritation, two important stages in the formation of plaque buildup, are triggered by ROS.

Obesity and Sedentary Lifestyle:
Increased fatty tissue, especially in the abdomen, induces high levels of insulin and inflammation-related cytokines. These effects are exacerbated by inactivity, which raises the susceptibility to cardiovascular problems.

Genetic Factors:
Atherosclerosis is predisposed by alterations in genes related to blood vessel function, swelling, and the breakdown of lipids. In particular, an alteration in the APOB gene can alter how cholesterol is metabolized.

Age and Gender:
As people age, their artery walls undergo functional and structural shifts that are marked by increased levels of oxidative stress and decreased flexibility. Women's risk increases after post menopause since they have declining estrogen concentrations, but men's risk increases throughout their youth owing to fluctuating hormones.

Epidemiology:
Cardiovascular illness is often regarded as "the disease of the century" considering it is the utmost prevalent cause of impairment and early death globally, with cardiovascular pathologies accounting for nearly half of deaths [13]. According to study findings, 422.7 million individuals had suffered from CVD as of 2020. More than 17 million deaths globally were lost due to CVD in 2015, making up about 31% of all fatalities worldwide. Although medical treatments are less accessible in countries with middle or low incomes, over 75% of deaths due to CVD generally occur there. This delays identification before the clinical period, increasing early death from CVD.[14] Conversely, wealthy countries have seen a rise in the prevalence and incidence of CVD resulting from lack of exercise, high levels of alcohol and smoking, and an imbalanced diet.

Pathogenesis:-
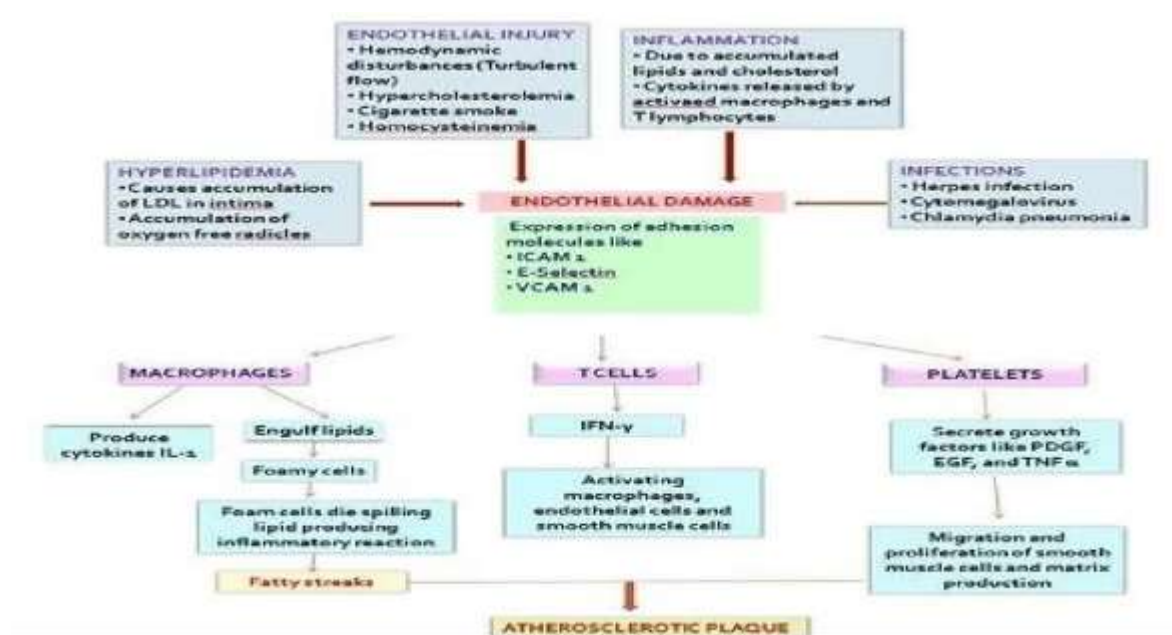
Risk factors:-
Occupational and psychological variables are examples of modifiable risk factors. A primary reason for this illness has been elevated lipids, namely low HDL and high LDL. High levels of blood pressure, or hypertension, weaken artery walls and increase their susceptibility to plaque accumulation. Smoking is another important consideration since it damages blood vessels and accelerates the accumulation of plaque. By raising blood glucose levels and intensifying inflammatory processes, hyperglycemia and resistance to insulin promote atherosclerosis. The risk is increased by excess weight, especially abdominal obesity, which is closely associated with lipid abnormalities and hypertension. Inadequate exercise and a diet high in sugar, fats, and saturated fats also have important effects. These fires will be fueled by ongoing stress and irregular sleep patterns, which raise blood pressure and inflammatory indicators. Additionally, high alcohol consumption can indirectly contribute to hypertension and lipid imbalance.

CLINICAL PRESENTATIONS:-

In its early stages, atherosclerosis frequently shows no symptoms and advances slowly. When plaque in the arteries ruptures or dramatically expands, it can cause blood clots or reduce blood flow. The arteries implicated and the extent of the blockage affect the clinical manifestations.Common presentations include Angina might feel like pressure or a tightness in your chest; atherosclerosis Shows no signs or symptoms or signs till it totally blocks an artery. It can occasionally be felt in the back, neck, jaw, shoulders, or arms. Usually, the discomfort gets greater as you move and goes away when you relax. The discomfort may also be triggered by emotional stress.

- Chest pain
- Nausea and vomiting sensation
- Fatigue
- Weakness
- Shortness of breath
- Depression and anxiety
- Heart attack
- Coronary thrombosis

**DIAGNOSIS**:-

Clinical Assessment:

To evaluate risk variables such as tobacco use, hypertension, diabetes, and family histories of coronary illnesses, an extensive medical history and physical assessment are crucial. Atherosclerosis risk may be indicated by decreased peripheral pulses, carotid bruits, or hypertension.

Blood Test: Lipid Profile:

High LDL, total, and triglyceride levels and low HDL cholesterol are significant markers of atherosclerosis.Extremely Sensitive High-sensitivity C-reactive protein (hs-CRP): Elevated levels of hs-CRP indicate systemic inflammation and an increased likelihood of cardiovascular events.Fasting Blood Glucose: Elevated blood sugar or aberrant glucose metabolism are frequently linked to atherosclerosis.

Imaging Techniques:

Ultrasound: Doppler ultrasonography of the lower limbs or carotid arteries can identify stenosis or plaque build-up. Carotid ultrasonography has a key role in detecting early atherosclerosis changes.CT stands for computed tomography. Angiography: A non-invasive imaging method that produces extremely high-resolution images of blood arteries and can identify coronary heart disease or peripheral artery disease.

Pharmacological Management:-

Anti-Hyperlipidemic Agents:
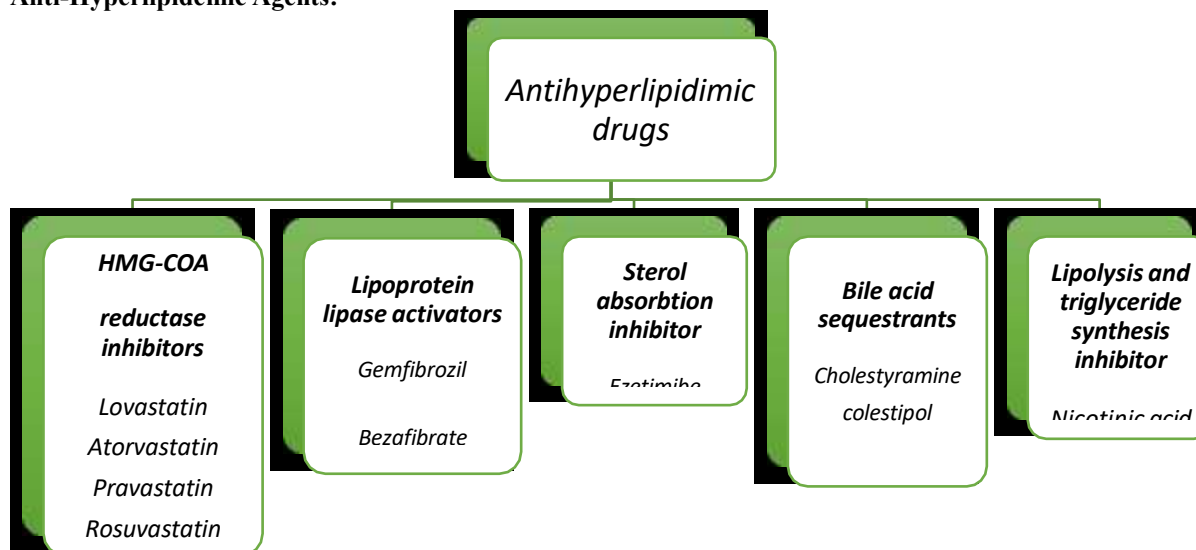
Classification of antihyperlipidemic drugs

Hmg-Coa Reductase Inhibitors:

Mechanism of Action:
The HMG-CoA reductase enzyme, which stimulates the conversion of HMG-CoA to mevalonate, the primary intermediary in the liver's synthesis of cholesterol, is inhibited by statins. This lowers intracellular cholesterol content and increases the activity of LDL receptors on the liver cell surface, which increases the removal of bad cholesterol from the bloodstream. As a result, statins lower triglycerides, LDL cholesterol, and total cholesterol while slightly increasing HDL cholesterol.

Dose: The dose varies depending on the specific statin and patient factors

- Atorvastatin   : 10–80 mg once daily
- Rosuvastatin   : 5–40 mg once daily
- Simvastatin   : 10–40 mg once daily (maximum 80 mg in specific cases)
- Pravastatin   : 10–40 mg once daily

Lipoprotein lipase activators:

Mechanism of Action:
The protein enzyme lipoprotein lipase (LPL) is mostly found on the surface of endothelial cells of capillaries in tissue such as skeletal muscle, the heart, and adipose tissue. LPL activators increase the activity of LPL. Triglycerides found in VLDL and chylomicrons are hydrolyzed by LPL to produce glycerol and free fatty acids. Tissues subsequently absorb these unbound fatty acids for deposition or energy generation. This procedure makes it easier to remove triglyceride-rich lipoproteins from the circulation and lowers the amount of triglycerides in the blood.

Dose:
Gemfibrozil : 600 mg twice daily, 30 minutes before meals.

Sterol absorption inhibitors:

Mechanism of Action:
The Niemann-Pick C1-Like 1 protein that is present on the brush border of the tiny intestine's wall is inhibited by ezetimibe. Its malfunction prevents the body from absorbing dietary and biliary cholesterol, which results in fewer lipids entering the liver. Reducing cholesterol in the liver promotes the development of additional LDL receptors, which increases the clearance of low-density lipoproteins (LDL) from the bloodstream.

Dose:
The usual dose of ezetimibe is 10 mg once daily, with or without food.

Bile acid sequestrants:

Mechanism of Action:
Resins that are incapable of being absorbed bile acid binders such as cholestyramine, colestipol, and colesevelam, function by binding bile acids within the intestines and preventing their release back into the enterohepatic circulation. Because of this decrease in bile acids, the liver synthesizes bile acids from cholesterol, which lowers the amount from cholesterol within cells. This causes the liver to produce more low-density lipoprotein (LDL) receptors, which increases the liver's absorption of LDL cholesterol from the bloodstream.

Dose:
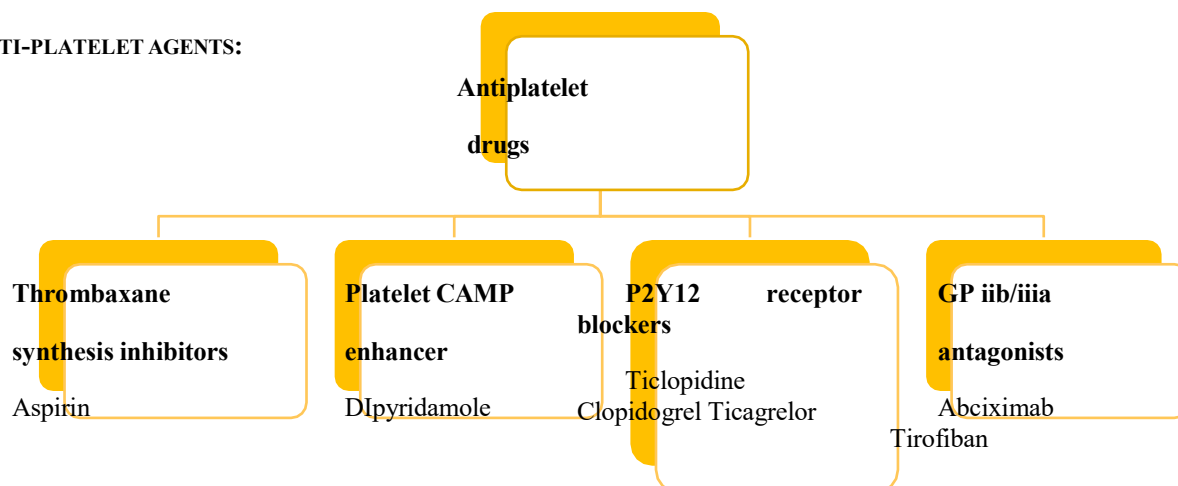Cholestyramine : 4–24 g daily, divided into 1–6 doses, mixed with water or juice.

Lipolysis and triglyceride synthesis inhibitors:

Mechanism of action:
Nicotinic acid reduces the action of lipolysis in stored fat by binding to G- protein-coupled receptors on fat cells, which results in an inhibition in the release of unbound fatty acids, even though it raises HDL cholesterol and decreases triglycerides and LDL cholesterol. It will result in a minor drop in VLDL synthesis and a decrease of hepatic triglyceride formation, which will consequently lower LDL levels. Moreover, by delaying the elimination of apolipoprotein A-I, niacin raises HDL contents.

Dose:
Initial dose: 250 mg daily, taken with food.
Maintenance dose: Gradually titrated to 1.5–2 g/day, divided into 2–3 doses.

ANTI-PLATELET AGENTS:

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                          ┌──────────────┐
                          │ Antiplatelet │
                          │    drugs     │
                          └──────┬───────┘
        ┌─────────────────┬──────┴──────┬──────────────────┐
┌───────────────┐ ┌──────────────┐ ┌──────────────┐ ┌──────────────┐
│ Thrombaxane   │ │ Platelet CAMP│ │ P2Y12        │ │ GP iib/iiia  │
│ synthesis     │ │ enhancer     │ │ receptor     │ │ antagonists  │
│ inhibitors    │ │              │ │ blockers     │ │              │
│               │ │ DIpyridamole │ │ Ticlopidine  │ │ Abciximab    │
│ Aspirin       │ │              │ │ Clopidogrel  │ │ Tirofiban    │
│               │ │              │ │ Ticagrelor   │ │              │
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Classification Of Anti-Platelet Drugs

Thromboxane Synthesis Inhibitors:

Mechanism of action:

Cyclooxygenase-1 in platelets is eternally inhibited by inhibitors of thromboxane A2 production, such as aspirin. Because of this inhibition, arachidonic acid cannot be converted into thromboxane A2, a potent vasoconstrictor that encourages platelet aggregation. This inhibition is irreversible by nature. For the full 7–10 day lifetime, platelets are unable to synthesize thromboxane A2.

Dose*:*
Aspirin is widely utilized for its blood-thinning properties at low doses (81–100 mg daily).
A loading dose of 150–300 mg, followed by 75–100 mg per day, is advised for acute coronary syndromes.

Platelet Camp Enhancers:
Mechanism of action:
By inhibiting the function of phosphodiesterase (PDE) enzymes or inducing adenosine monophosphate synthesis by adenylyl cyclase, platelet cAMP enhancers, like dipyridamole, increase cyclic adenosine monophosphate (cAMP) levels. Platelet stimulation and aggregation are decreased due to the elevated cAMP levels lowering intracellular calcium levels. Additionally, dipyridamole prevents adenosine from being reabsorbed, thereby raising cAMP levels.

Dose:
In combination with aspirin, take 75–100 mg of dipyridamole four times a day or 200 mg twice a day.
Cilostazol: 100 mg twice a day for vasodilatory and antiplatelet effects.

P2y12 Receptor Blockers:

Mechanism of action:

Clopidogrel, ticagrelor, and prasugrel are examples of P2Y12 inhibitors that block the platelets' P2Y12 receptor. This receptor is necessary for the ADP-induced activation and aggregation of platelets. By preventing platelet aggregation or cross-linking, blockage therefore offers a way to suppress activation and, hence, thrombus development.

Dose:
Clopidogrel: 75 mg every day after a loading dose of 300–600 mg.
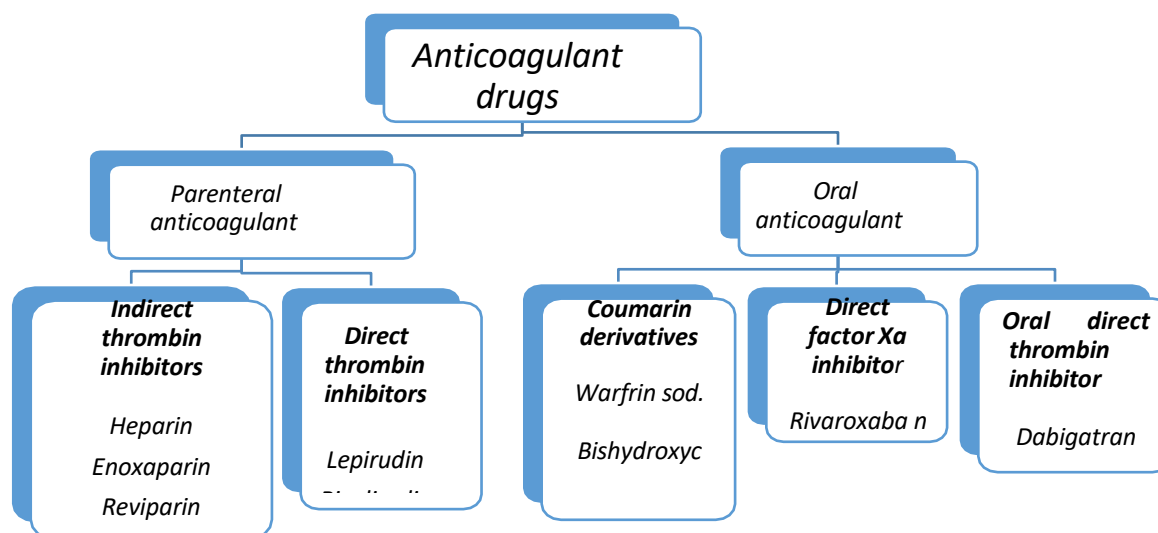Prasugrel: 10 mg per day after a 60 mg loading dosage.
Ticagrelor: 90 mg twice a day after a loading dose of 180 mg.

Glycoprotein iib/iiia antagonists:

Mechanism of action:
GPIIb/IIIa antagonists, such as tirofiban, eptifibatide, and abciximab, occupy platelet GPIIb/IIIa receptors and serve as the main pathway for platelet clumping. These medications prevent platelet aggregation by preventing von Willebrand factor and fibrinogen from attaching to the receptor.

Dose:
Abciximab: an IV bolus of 0.25 mg/kg, followed by a 12-hour infusion of 0.125 mcg/kg/min.

ANTICOAGULANTS:

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                        Anticoagulant
                            drugs
                   ┌──────────┴──────────┐
              Parenteral                Oral
             anticoagulant          anticoagulant
           ┌──────┴──────┐      ┌────────┼────────┐
       Indirect      Direct  Coumarin   Direct   Oral direct
       thrombin     thrombin derivatives factor Xa thrombin
       inhibitors   inhibitors           inhibitor inhibitor
                              Warfrin sod.
       Heparin      Lepirudin           Rivaroxaba n
                              Bishydroxyc          Dabigatran
       Enoxaparin
       Reviparin
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Classification Of Anticoagulant Drugs

Indirect Thrombin Inhibitors:

Mechanism of action:
 Indirect thrombin inhibitors, namely low molecular weight heparins (LMWHs), including enoxaparin and unfractionated heparin (UFH), boost the function of antithrombin III. This interaction leads to the inactivation of factor Xa and thrombin, also known as factor IIa. While UFH suppresses both thrombin and factor Xa activity, LMWH inhibits a significant portion of factor Xa's activity due to its shorter molecular chain.
Dose:
Enoxaparin:
- 40 mg given via subcutaneous injection once per day for prophylaxis.
- Subcutaneous dosage of 1 mg per kg every 12 hours is the treatment.

DIRECT THROMBIN INHIBITORS:

Mechanism of action:
The DTI medications are argatroban, bivalirudin, and dabigatran. DTI interacts primarily with factor IIa, the thrombin sites of action. As a result, they inhibit fibrinogen's thrombin-dependent cleavage into fibrin. By blocking the connection with fibrinogen and thrombin, clotting becomes less prevalent. Antithrombin III is not involved in DTI's actions.

Dose:
Dabigatran: In atrial fibrillation, 150 mg twice daily is recommended for stroke prevention (reduce to 75 mg twice daily in renal impairment).
Bivalirudin: An infusion of 1.75 mg/kg/hour following an initial intravenous bolus of 0.75 mg/kg for the purpose of percutaneous coronary intervention.

Coumarin Derivatives:
Mechanism of action:
Vitamin K epoxide reductase, or VKORC1, is inhibited by warfarin as well as coumarin derivatives. Thus, it decreases the process of active vitamin K and prevents clotting factors II, VII, IX, and X, along with proteins C and S, from being carboxylated. The anticoagulant impact of active clotting factors is delayed because the body takes longer to deplete them.

Dose:
For the first 1-2 days, the initial dosage is 5-10 mg taken orally once day, it is then increased to maintain an INR of 2-3 (for the majority of reasons).

Direct Factor Xa Inhibitors:

Mechanism of action:
Prothrombin production is prevented by direct factor Xa inhibitors such as rivaroxaban, apixaban, and edoxaban, which block factor Xa from limiting it. Consequently, it prevents both the clots and the formation of thrombin.

Dose:
Rivaroxaban : 15 mg twice day for 21 days, followed by 20 mg once daily for pulmonary embolism (PE) and deep vein thrombosis (DVT).
Apixaban : 5 mg twice daily (down to 2.5 mg twice daily under certain circumstances) is the advised dosage for atrial fibrillation stroke prevention.

NON PHARMACOLOGICAL MANAGEMENT:
Nutritional Measures:
Consuming red meat and full-fat dairy products includes saturated fats, which increase LDL cholesterol levels. Healthy fats, among those in nuts, fish, and olive oil, may be utilized to reduce cholesterol levels. Consuming excessive amounts of salt leads to high blood pressure, which is the largest risk factor for atherosclerosis. Cutting less salt intake can help lower blood vessel strain and regulate blood pressure.

Physical Activity:
Walking, cycling, and swimming are examples of aerobic exercises that can raise HDL, or good, cholesterol while lowering LDL and triglyceride levels. By reducing both the diastolic and systolic readings, exercise decreases blood pressure. As a result, there is reduced stress on the arteries, which reduces the risk of plaque rupture and clot formation.

Smoking Quitting:
When quitting smoking, endothelial function enhances vasodilation and overall circulatory function. Smoking constitutes among the most significant underlying causes of cardiovascular disease. Giving up this dangerous behaviour reduces the risk of cardiovascular incidents, including coronary heart disease and stroke that are linked to atherosclerosis.

SURGICAL MANAGEMENT:
**Coronary Artery Bypass Grafting, Or Cabg:**
CABG is a form of surgery that employs grafting from various body areas, including the internal mammary artery or saphenous vein, to bypass blocked coronary arteries. By providing a different path for the flow of blood, CABG greatly enhances the supply of oxygen that gets to the heart muscle. Patients with diabetes, left main heart disease, or multi-vessel disease can benefit greatly from it. Major coronary artery disease can be effectively treated with this operation over the long term, which lowers symptoms and increases survival.

Percutaneous Transluminal Coronary Angioplasty (Ptca):
The minimally invasive procedure known as percutaneous transluminal coronary angioplasty (PTCA) involves inserting a balloon catheter into the constricted artery. When inflated, this balloon promotes blood flow by stretching the artery. PTCA is the best treatment for localized artery blockages and is frequently performed in conjunction with stent insertion. It is perfect for individuals who may not be able to handle open heart surgery and has a quicker recovery period.

Intra-Aortic Balloon Pump (Iabp):
When severe cardiac failure, including cardiogenic shock, is occurring in a patient, the Intra-Aortic Balloon Pump (IABP) must be used. By inflating during the diastolic phase and deflating during the systolic phase, the approach increases coronary artery perfusion and reduces left ventricular workload. IABP is widely utilized as a bridge to final management treatments, most notably CABG or heart transplant, even if it has remained a "supportive only" therapy approach.
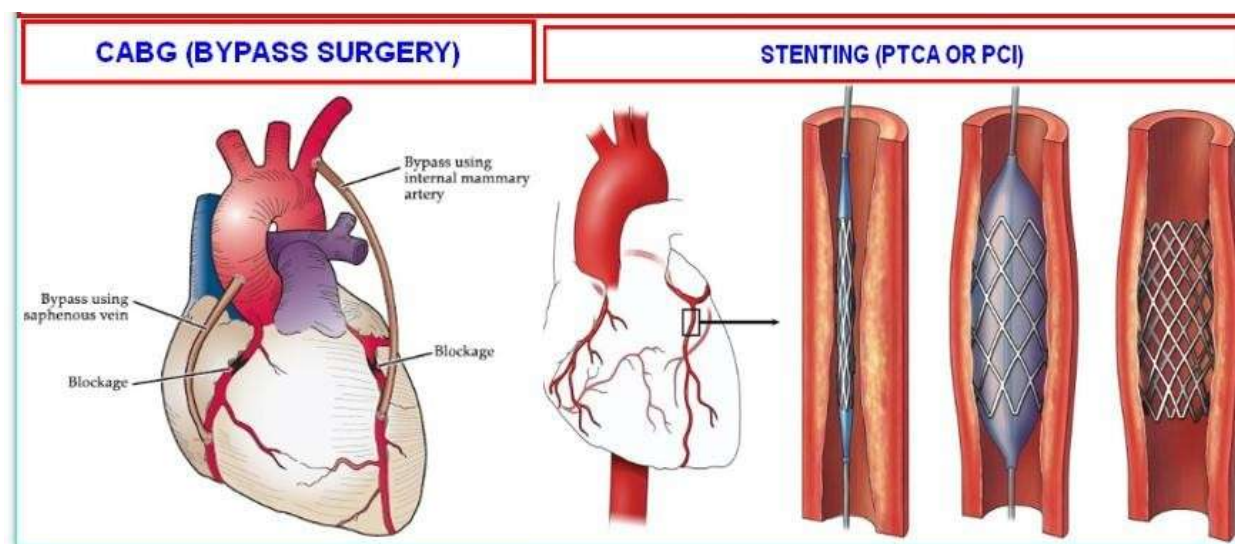
Cabg Vs. Stenting: Coronary Artery Treatment Options

METHODOLOGY MATERIALS AND METHODS

STUDY DESIGN	PROSPECTIVE OBSERVATIONAL STUDY
SAMPLE SIZE	100
STUDY SITE	MULTI SPEACIALITY HOSPITAL
DEPARTMENT	CARDIOLOGY
STUDY DURATION	6 MONTHS

Source Of Data and Materials:

Patients consent forms
Lab reports / angiogram report
Patient data collection form
Patients prescription

Inclusion criteria:

Age above 18 years.
Patients with cardiovascular disease / atherosclerosis
Patients who are willing to participate in the study

Exclusion criteria:

Patients with impairments
Pregnant women , lactating
Patients below the age of 18

Study procedure:

All eligible patients diagnosed with atherosclerosis based on clinical evaluation and confirmed through angiographic reports were enrolled after obtaining informed consent. Baseline data including demographic details, medical history, lifestyle factors, and current medications were recorded. Angiogram reports were thoroughly assessed to evaluate the location, severity, and extent of arterial stenosis.

Patients were then managed according to standard therapeutic strategies, including pharmacological treatments such as statins, antiplatelets, antihypertensives, and antidiabetic agents, along with non-pharmacological approaches like lifestyle modifications.

RESULTS:-

Distribution Of Patients Based On Sex

DISTRIBUTION OF PATIENTS BASED ON SEX

28%

72%
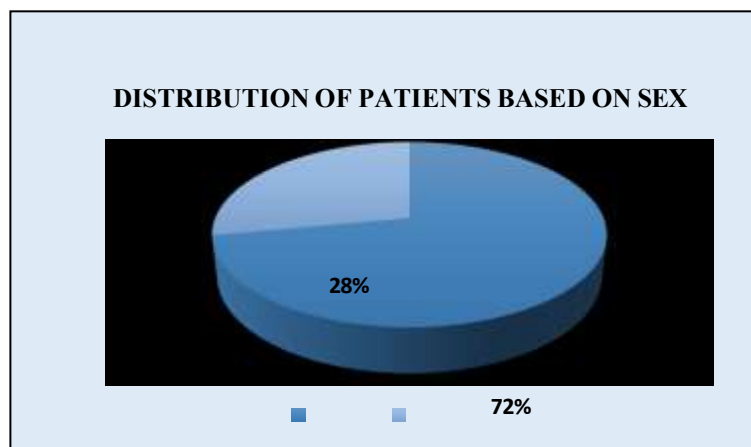
Here it was observed that based on sex 72 patients were male and 28 patients were female.

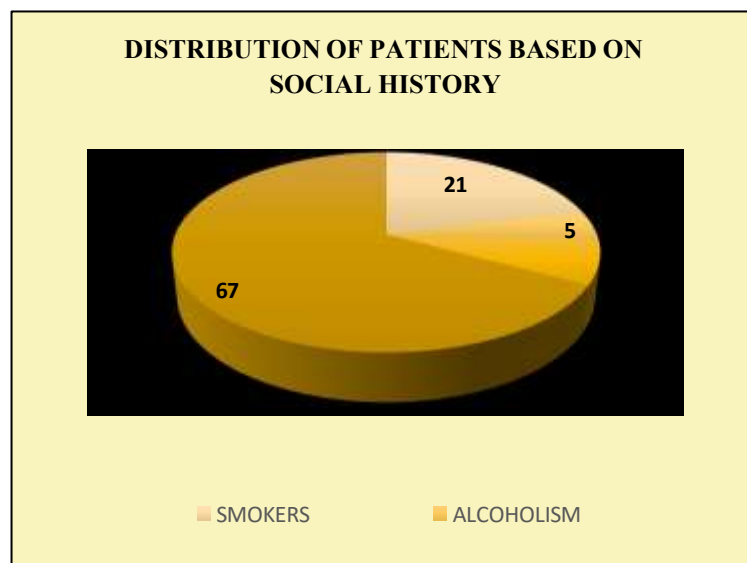DISTRIBUTION OF PATIENTS BASED ON AGE

DISTRIBUTION OF PATIENTS BASED ON AGE

MALE   FEMALE
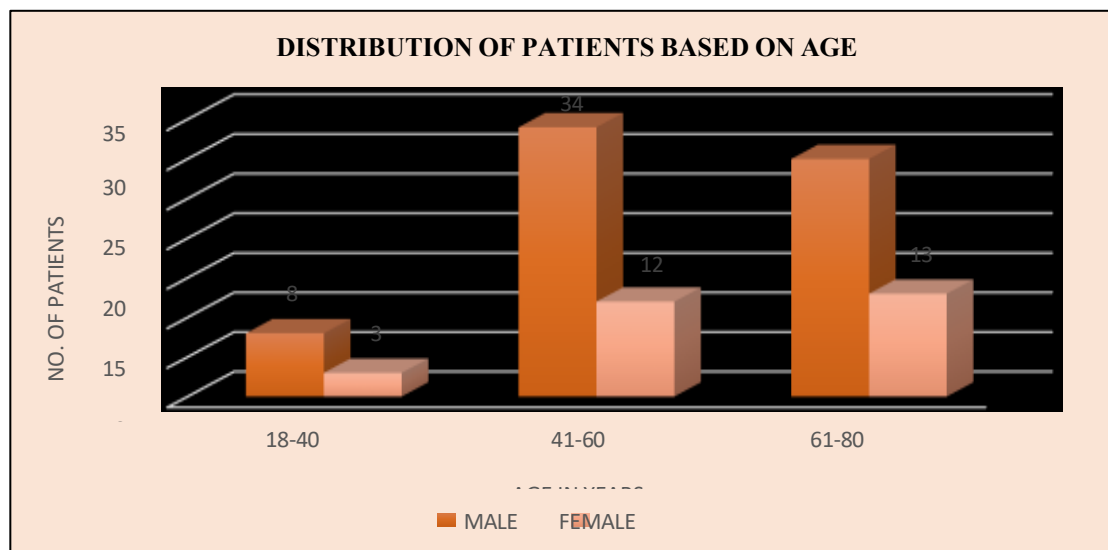
Atherosclerosis in the age of 18-40 years males were found to be 8 and females were 3, 41-60 years males were 34 and female were 12 and at the age of 61-80 males were 30 and females were 13.

DISTRIBUTION OF PATIENTS BASED ON OCCUPATIONAL STATUS

DISTRIBUTION OF PATIENTS BASED ON OCCUPATIONAL STATUS

NO. OF PATIENTS

OCCUPATION OF THE PATIENTS
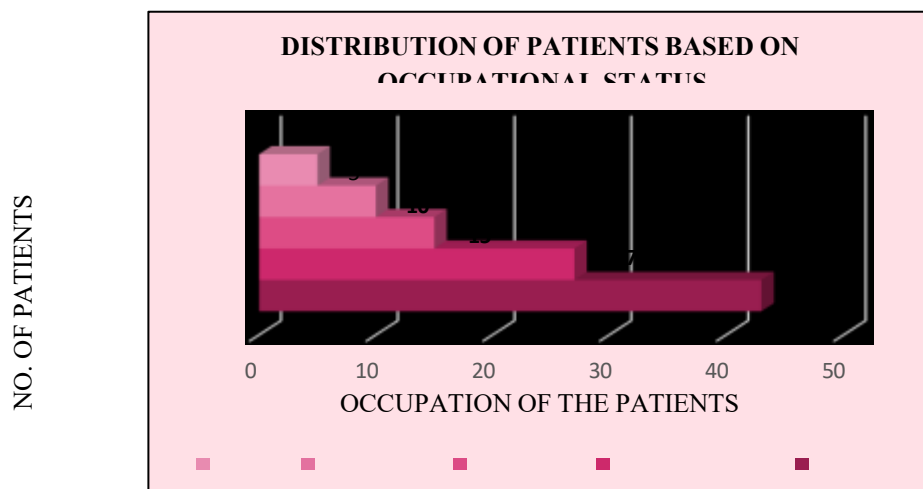
Here we observed that distribution of disease in the patients based on their occupational status in which drivers were found 43, 27 were office workers, labourers were 15, 10 were house wife and 5 were others.

**DISTRIBUTION OF PATIENTS BASED ON SOCIAL HISTORY**

Patients with the social history i.e., smokers were found to be 21, 5 patients with alcoholism, 7 patients were tobacco chewers and 67 patients were with no social history.
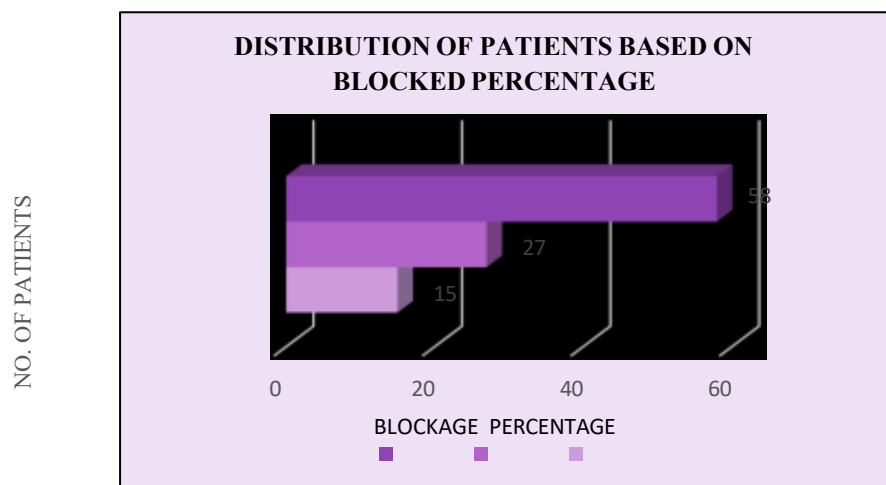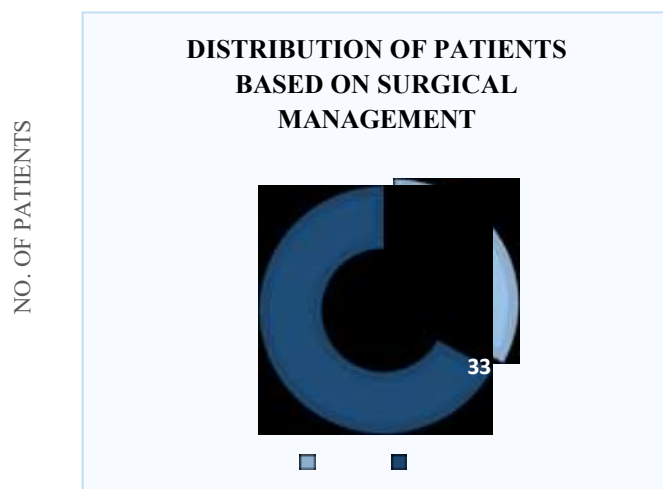
DISTRIBUTION OF PATIENTS BASED ON MEDICAL HISTORY
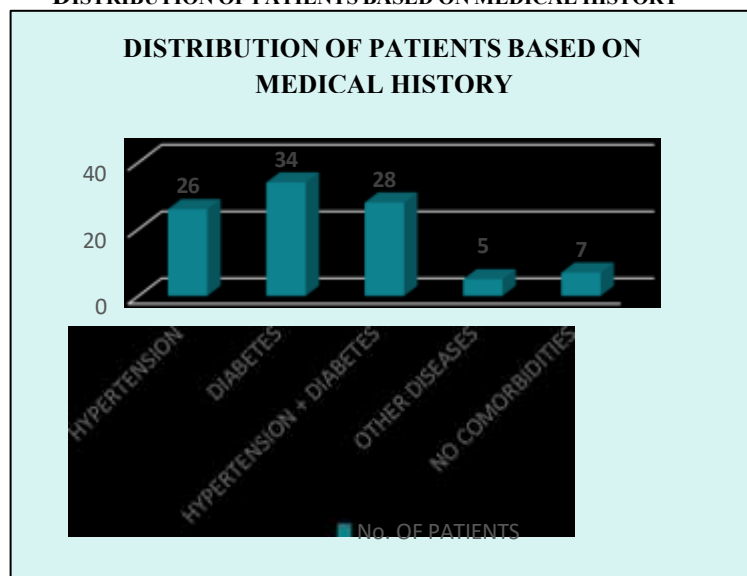
Patients with comorbid conditions such as Hypertension were found to be in 26 patients, 34 patients were diabetic, 28 patients were found in combine comorbid condition i.e., Hypertension+ Diabetes, 5 patients with other diseases and 7 patients were with no comorbid conditions.

DISTRIBUTION OF PATIENTS BASED ON ETIOLOGICAL DIAGNOSIS
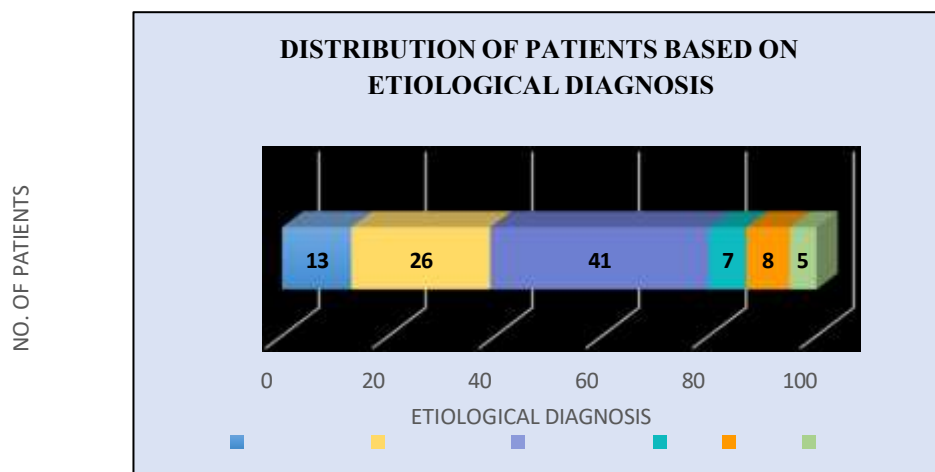
CAD-SVD was diagnosed in 13 patients, 26 patients were diagnosed with CAD-DVD, 41 patients were diagnosed with CAD-TVD, 7 patients diagnosed with MI, 8 patients diagnosed with ACS and 5 patients were diagnosed with PAD.
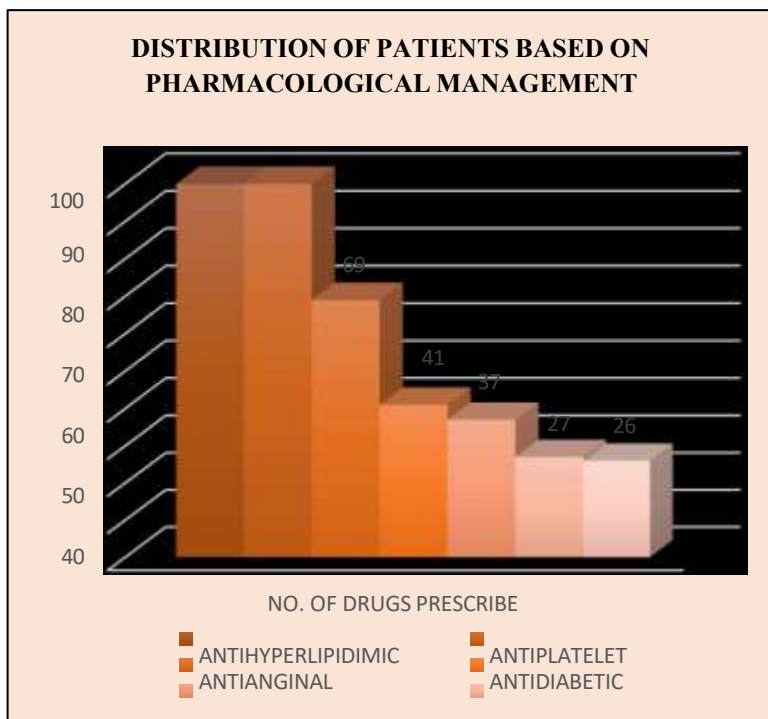
DISTRIBUTION OF PATIENTS BASED ON BLOCKED PERCENTAGE

Here we observe that 10-30% of blockage was found in 15 patients, 27 patients had 31-60% blockage, 61-90% of blockage was found in 58 patients.
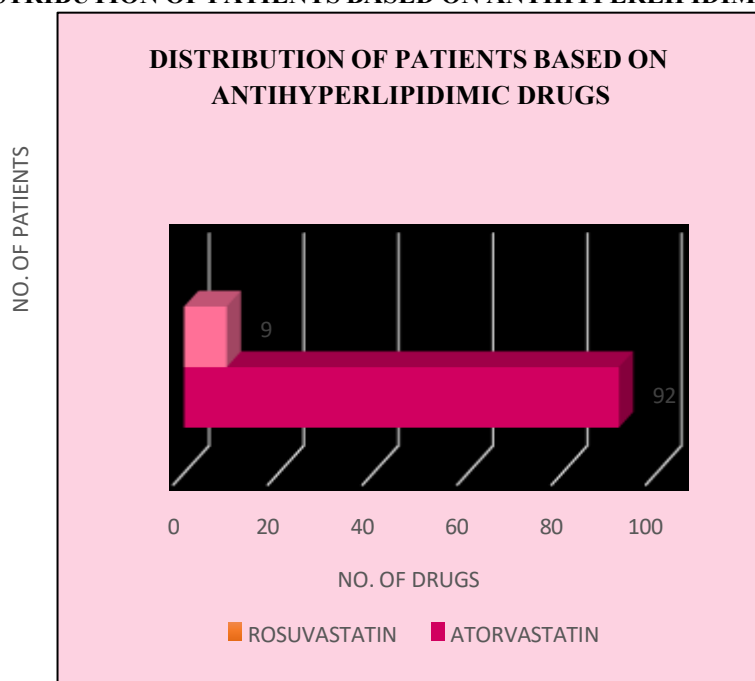
DISTRIBUTION OF PATIENTS BASED ON SURGICAL MANAGEMENT

Here we observe that 33 patients were undergone for CABG and 67 patients were suggested for PTCA surgical management.
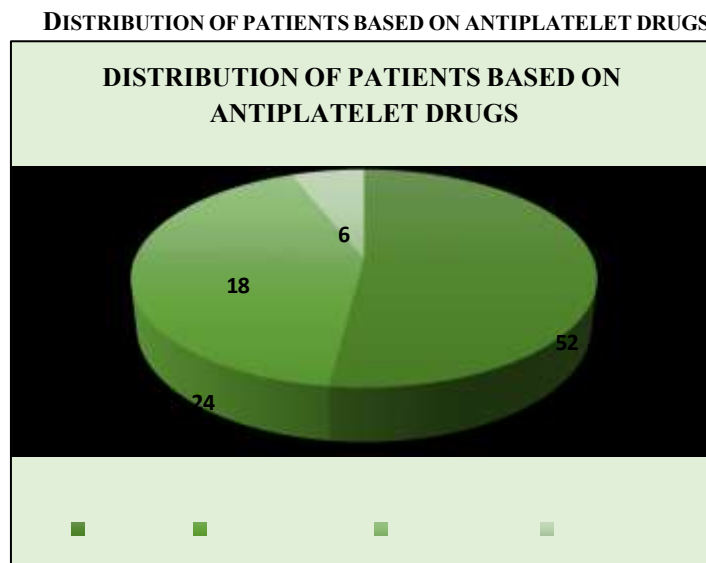
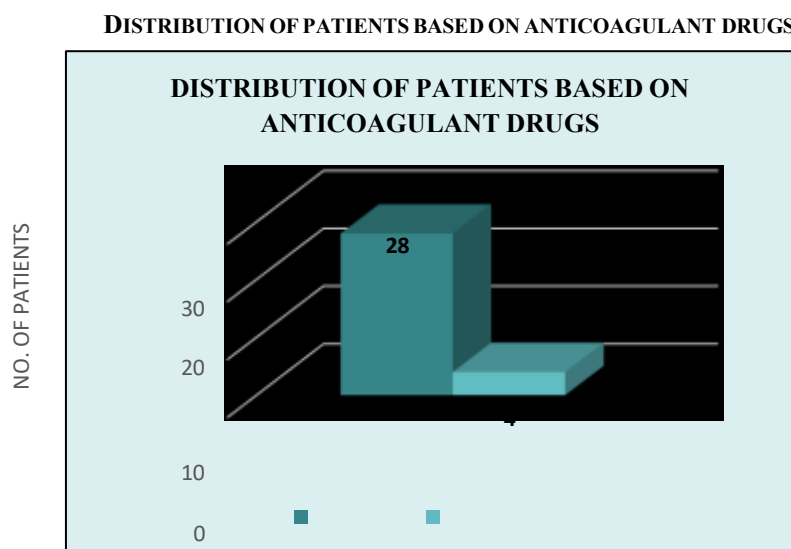**DISTRIBUTION OF PATIENTS BASED ON PHARMACOLOGICAL MANAGEMENT**

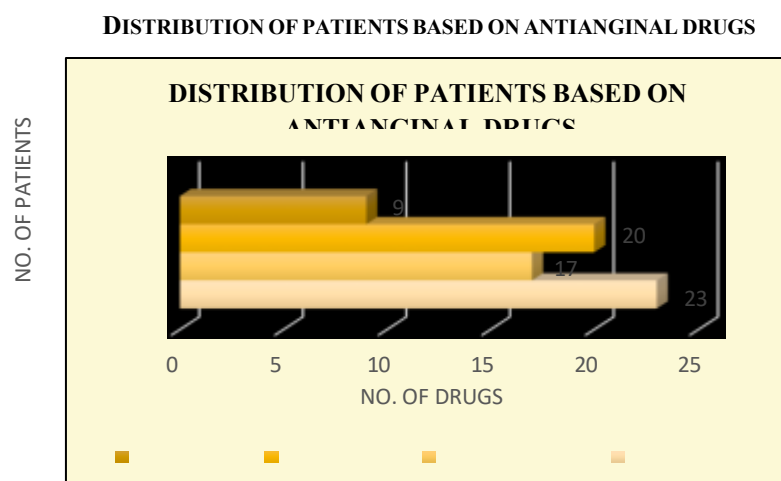**DISTRIBUTION OF PATIENTS BASED ON ANTIHYPERLIPIDIMIC DRUGS**

Atorvastatin was prescribed in 92 patients; 8 patients were prescribed Rosuvastatin.

DISTRIBUTION OF PATIENTS BASED ON ANTIPLATELET DRUGS

Aspirin was prescribed in 52 patients, 24 patients were prescribed clopidogrel, 18 patients were prescribed ticagrelor and tirofiban was prescribed in 6 patients.
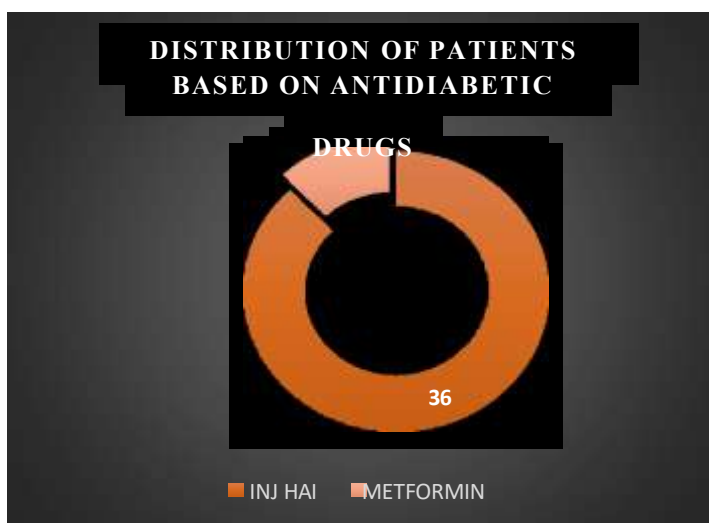
DISTRIBUTION OF PATIENTS BASED ON ANTICOAGULANT DRUGS

It was observed that heparin was prescribed in 28 patients and 4 patients were prescribed enoxaparin.

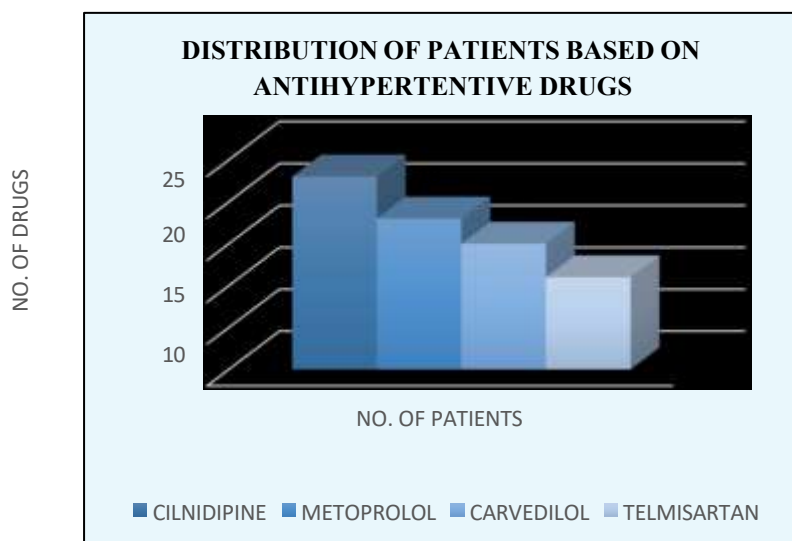DISTRIBUTION OF PATIENTS BASED ON ANTIANGINAL DRUGS

Nicorandil was prescribed in 23 patients, 17 patients were prescribed nitroglycerin, 20 were prescribed ranolazine, and isosorbide was prescribed in 9 patients.

**DISTRIBUTION OF PATIENTS BASED ON ANTIDIABETIC DRUGS**

DISTRIBUTION OF PATIENTS BASED ON ANTIDIABETIC DRUGS

36

■ INJ HAI   ■ METFORMIN

Inj. HAI was given in 36 patients, 5 patients were given metformin.

DISTRIBUTION OF PATIENTS BASED ON ANTIHYPERTENTIVE DRUGS

DISTRIBUTION OF PATIENTS BASED ON ANTIHYPERTENTIVE DRUGS

NO. OF DRUGS

NO. OF PATIENTS

■ CILNIDIPINE ■ METOPROLOL ■ CARVEDILOL ■ TELMISARTAN

Cilnidipine was prescribed to 23 patients, 18 patients were given metoprolol, 15 patients were given carvedilol, telmisartan was prescribed in 11 patients.

DISTRIBUTION OF PATIENTS BASED ON DIURETICS

DISTRIBUTION OF PATIENTS BASED ON DIURETICS

NO. OF PATIENTS

14   8   5

0%   20%   40%   60%   80%   100%

NO. OF DRUGS
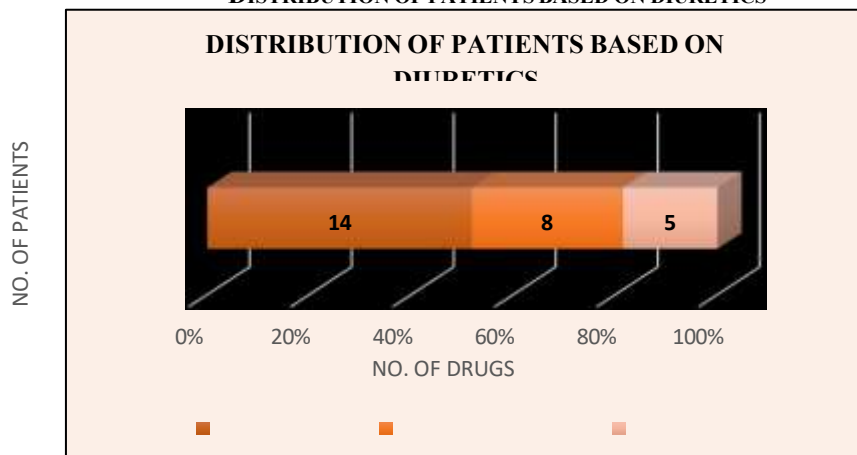
Furosemide was given in 14 patients, 8 patients were given spironolactone, torsemide was prescribed in 5 patients.

Discussion:-

In our study, we observed that antihyperlipidemics and antiplatelets were commonly prescribed for the therapeutic management of atherosclerosis and other related disorders. Furthermore, the majority of the patients included in our study were male, over 41-80 years old, indicating that atherosclerosis is most commonly seen in elderly patients.

Atorvastatin was the drug most often prescribed to patients, followed by aspirin. The most commonly prescribed statin was atorvastatin under the brand Atorvas, and the next statin commonly prescribed was rosuvastatin, available as Rosuvas.

Coronary angiography and 2D echocardiography were commonly used to diagnose CAD, while angiography was used to diagnose PAD. An abnormal ECG was used to diagnose ACS.

The most common occupational status in our study is of drivers, followed by office workers, followed by labourers, followed by house wife, followed by others.

The most common social history in our study is of smokers, followed by tobacco chewers, followed by alcoholism, followed by no social history.

The most common etiological diagnosis in our study is CAD TVD followed by CAD - DVD, followed by CAD – SVD , followed by ACS , followed by MI , followed by PAD The most common surgical procedure in our study is PTCA followed by CABG

The most commonly given antiplatelet is aspirin (brand name Ecospirin), followed by clopidogrel (brand name Clopitab).The most often given anticoagulant is heparin (brand name Heparin), followed by enoxaparin.The most commonly prescribed antianginal medicine is nicorandil (Nikoran), followed by ranolazine (Rancad).

Diabetes mellitus was the most common comorbidity observed in our subject, followed by hypertension. The study included 100 participants with coronary artery disease, acute coronary syndrome, myocardial infarction, and peripheral artery disease. The most common complaints included chest pain, shortness of breath, palpitations, and back pain.

Requirements For Future Drug Development:
Addressing Residual Risk:
There is still a significant amount of residual risk even if current medications like statins and antiplatelet treatments have significantly decreased cardiovascular events. These new treatments ought to concentrate on processes that have not previously been addressed, such as chronic inflammation, which plays a crucial role in the advancement of atherosclerosis. Novel anti- inflammatory drugs that target NLRP3 or interleukin-6 inflammasomes will prove to be very successful. Lipoprotein, a genetically determined, hereditary lipoprotein linked to cardiovascular risk, is another possibility that is gaining interest. Treatments that target RNA-based inhibitors are being researched to lower levels of lipoprotein.

Enhanced Safety Profiles:
Safety should come first while developing treatments for atherosclerosis. In fact, even though there isn't any concrete proof of muscle injury, a significant number of patients discontinue statins due to adverse effects. To lessen these problems, different medications, such as bempedoic acid, are being developed as safer substitutes or supplements to statins. Although they can also be quite successful, anticoagulants and antithrombotics sadly have a bleeding risk that must be properly handled for high-risk or older groups. Along that horizon, there will be advancements in medication formulations, precision dosage, and drugs like factor XI inhibitors that have a decreased risk of bleeding

Cost-Effectiveness and Accessibility:
Because of their historically high prices, modern medicines like PCSK9 inhibitors and monoclonal antibodies are not as widely available worldwide. The creation of biosimilars and small-molecule substitutes may often lower production costs while producing the same result. Other viable alternatives may entail public-private cooperation that results in the development of pricing structures that guarantee reasonably priced pharmaceuticals. Fairness and equity in access will also be greatly aided by grassroots education and initiatives to upgrade the healthcare system in low- and middle-income (LMIC) nations.

Long-Term Efficacy Studies:
Drug approval procedures nowadays might not sufficiently consider the long-term impacts of medications as they are primarily focused on gauging short- to medium-term results. In order to evaluate treatment durability and identify late-emerging adverse events, future studies must concentrate on extended follow-up periods. AI and the implementation of electronic medical records should guarantee the gathering of empirical data after approval. This approach will ensure that treatments continue to be safe and effective across a range of clinical situations and demographics.

CONCLUSION:-
The goal of our prospective observational study is to evaluate the therapeutic strategies in the management of atherosclerosis with different class of drugs. The therapeutic strategies include the use of antihyperlipidmic agents such as atorvastatin which reduces low density lipoprotein (LDL). Antiplatelet medications are used to prevent the sticking of platelets together and forming clumps which leads to formation of clots. Anticoagulants such as aspirin and heparin also being used to prevent thrombotic events.

However, since this was a prospective observational study, no interventions were made. Most of the patients were found to be with coronary artery disease followed by myocardial infraction and acute coronary syndrome. The patients with coronary artery disease was treated with aspirin and clopidogrel and patients with acute coronary syndrome was treated with statin and antiplatelet therapies.

Antianginal drugs such as ranolazine and nicorandil are prescribed based on symptoms. Optimizing treatment through personalized therapy and regular monitoring improves patient outcomes and lowers cardiovascular events. The data show that atorvastatin + aspirin + nicorandil is the most commonly prescribed combination of pharmacological therapy in atherosclerosis.

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Bibliography:-
1. Libby, P., Ridker, P. M., & Hansson, G. K. (2011) 24(6), 515–523.
2. Ross, R. Atherosclerosis an inflammatory disease. New England Journal of Medicine, (1999). 340(2), 115-126.
3. World Health Organization. Cardiovascular diseases (CVDs). Retrieved from WHO website. (2021).
4. Benjamin, E. J., et al. Heart Disease and Stroke StatisticsUpdate. Circulation, (2019) 139(10), e56–e528.
5. Frostegård, J. Immunity, atherosclerosis and cardiovascular disease. BMC Medicine, (2013). 11(1), 117.
6. Beckman, J. A., Creager, M. A., & Libby, P. Diabetes and atherosclerosis: epidemiology, pathophysiology, and management. JAMA, (2002). 287(19), 2570–2581.
7. Tabas, I., Williams, K. J., & Boren, J. Subendothelial lipoprotein retention as the initiating process in atherosclerosis. Circulation, (2015). 116(16), 1832–1844.
8. Ambrose, J. A., & Barua, R. S.The pathophysiology of cigarette smoking and cardiovascular disease: An update. Journal of the American College of Cardiology, (2004). 43 (10), 1731–1737.
9. Madamanchi, N. R., Vendrov, A., & Runge, M. S. Oxidative stress and vascular disease. Arteriosclerosis, Thrombosis, and Vascular Biology, (2005). 25(1), 29–38.
10. Katsiki, N., Perez-Martinez, P., & Mikhailidis, D. P. Obesity, diabetes, and vascular diseases: Lessons from the COVID-19 pandemic. Current Vascular Pharmacology, (2019). 17(4), 345–352.
11. Schunkert, H., et al. Large-scale association analysis identifies 13 new susceptibility loci for coronary artery disease. Nature Genetics, (2011). 43(4), 333–338.
12. North, B. J., & Sinclair, D. A.The intersection between aging and cardiovascular disease. Circulation Research, (2012). 110(8), 1097–1108.
13. Burlutskaya, A.V.; Tril, V.E.; Polischuk, L.V.; Pokrovskii, V.M. Dyslipidemia in pediatrician's practice. Rev. Cardiovasc. Med. 2021, 22, 817–834. [CrossRef]
14. Mozaffarian, D. Global scourge of cardiovascular disease: Time for health care systems reform and precision population health. J. Am. Coll. Cardiol. 2017, 70, 26–28. [CrossRef]
15. Mathers, C.D.; Loncar, D. Projections of global mortality and burden of disease from 2002 to 2030. PLoS Med. 2006, 3, e442. [CrossRef]

16. Hong, Y.M. Atherosclerotic cardiovascular disease beginning in childhood. Korean Circ. J. 2010, 40, 1–9. [CrossRef] [PubMed]

17. Mainieri, F.; La Bella, S.; Chiarelli, F. Hyperlipidemia and Cardiovascular Risk in Children and Adolescents. Biomedicines 2023, 11, 809. [CrossRef] [PubMed]

18. Touboul, P.J.; Hennerici, M.G.; Meairs, S.; Adams, H.; Amarenco, P.; Bornstein, N.; Csiba, L.; Desvarieux, M.; Ebrahim, S.; Woo, K.S.; et al. Mannheim carotid intima-media thickness and plaque consensus (2004–2006–2011). An update on behalf of the advisory board of the 3rd, 4th and 5th Watching the Risk Symposia, at the 13th, 15th and 20th European Stroke Conferences, Mannheim, Germany, 2004, Brussels, Belgium, 2006, and Hamburg, Germany, 2011. Cerebrovasc. Dis. 2012, 34, 290–296. [PubMed]

19. O'Leary, D.H.; Bots, M.L. Imaging of atherosclerosis: Carotid intima-media thickness. Eur. Heart. J. 2010, 31, 1682–1689. [CrossRef] [PubMed]

20. Enos, W.F.; Holmes, R.H.; Beyer, J. Coronary disease among United States soldiers killed in action in Korea; preliminary report. J. Am. Med. Assoc. 1953, 152, 1090–1093. [CrossRef] [PubMed]

21. Hansson, G. K., & Hermansson, A. The immune system in atherosclerosis. Nature Immunology, (2011). 12(3), 204–212.

22. Moore, K. J., & Tabas, I. Macrophages in the pathogenesis of atherosclerosis. Cell, (2011) 145(3), 341–355.

23. Bennett, M. R., Sinha, S., & Owens, G. K. Vascular smooth muscle cells in atherosclerosis. Circulation Research, (2016). 118(4), 692–702.

24. Virmani, R., et al. Pathology of the vulnerable plaque. Journal of the American College of Cardiology, (2005). 47(8S), C13–C18.

25. Hansson, G. K.Inflammation, atherosclerosis, and coronary artery disease. New England Journal of Medicine, (2005) 352(16), 1685–1695.

26. Libby, P., & Hansson, G. K. Inflammation and atherosclerosis: From pathogenesis to therapeutic targeting. Journal of the American College of Cardiology, (2015). 65(17), 1577-1589.

27. Benjamin, E. J., et al. Heart disease and stroke statistics—2019 update. Circulation, 139(10), e56-e528. World Health Organization (WHO). (2021). Cardiovascular diseases (CVDs) fact sheet. (2019).

28. Ridker, P. M., et al. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. New England Journal of Medicine, 359(21), 2110-2120. (2011).

29. Luo, X., et al. Carotid ultrasound for the diagnosis and assessment of atherosclerosis. Vascular Medicine, (2019) 24(6), 515–523.

30. Mahnken, A. H., et al. CT angiography in coronary artery disease: Current state of the art. European Heart Journal, (2008)29(7), 843-851.

31. Cohen AT et al., "Direct Oral Anticoagulants," European Heart Journal, 2016.

32. Nathan DM et al., "Management of Hyperglycemia in Type 2 Diabetes," Diabetes Care, 2022.