APROSPECTIVEOBSERVATIONALSTUDYONTHEEVALUATION OF THERAPEUTIC STRATEGIES IN THE MANAGEMENT OF ATHEROSCLEROSIS: A COMPHREHENSIVE CLINICAL AND PHARMACOLOGICAL APPROACH

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ABSTRACT

BACKGROUND: The goal of our study was to evaluate the comprehensive analysis of prospective studyofthe therapeutic management of Atherosclerosis. Arteriosclerosis is a type of vascular disease where the blood vessels carrying oxygen away fromthe 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 onclinicaland pharmacologicalroles ofantihyperlipidimic, antiplatelet, anticoagulant, antianginal, antidiabetic, antihypertensive and diuretics in the disease progression and patient outcome

METHODOLOGY: It was hospitalbased comprehensive studyconducted byrandomselection of patients. A prospective observational study involving analysis of inpatients of cardiology departmentin multispeacialityhospital. Alleligiblepatientsdiagnosedwithatherosclerosisbasedonclinical evaluation and confirmed through angiographic reports were enrolled after obtaining informed consent. Baselinedataincludingdemographicdetails, medicalhistory, lifestyle factors,andcurrent medicationswererecorded.Angiogramreportswerethoroughlyassessedtoevaluatethelocation, 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 totalof100 cases ofatherosclerosis were observed. Majorityofcases were males (72%) compare to females (28%). The most often prescribed antihyperlipidimic is Atorvastatin (92%)comparetorosuvastatin(8%).ThemostfrequentlyprescribedantiplateletisAspirin(52%) comparetoclopidogril(24%).Mostcommonlyprescribedanticoagulantisheparin(28%)compare toenoxaparin(4%).Themost commonsocialhistoryisofSmokers(21%), followedbyTobacco

chewers(7%) , alcoholism (5%) , followed by no social history (67%). The most common eliologicaldiagnonsisofCADTVD(77%)followedbyCAD -DVD(26%),followedbyCAD - SVD (13%) , followed by ACS (8%) , followed by MI (7%) , followed byPAD (5%) .

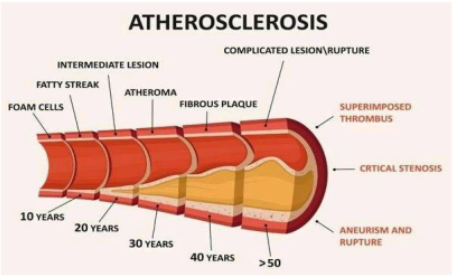
CONCLUSION: It is clearly seen from results that Atorvastatin + Aspirin + Nicorandil is the most prominent drug combination. These were also drug of choice at the place of study given majority of patients during course of our study other classes of drugs such as Rosuvastatin and tirofiban which were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

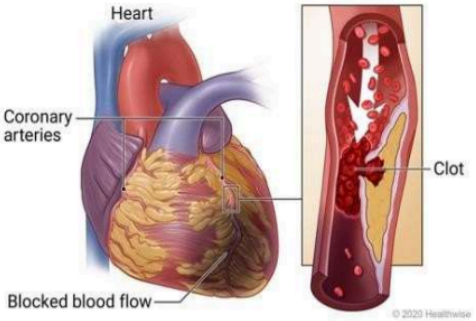
KEYWORDS: Atherosclerosis, Coronary artery disease, Antihyperlipidimic, Antiplatelet, Anticoagulant, Angiogram, Acute coronary syndrome, Inflammation, plaques, lipids.

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 narrowandbecomelesselastic.Plaquesaremadeoffibrousmaterialcontaininglipids,cholesterol, calcium,andcellularwaste.Theseincludethecoronary,carotid,andperipheralarteries,whichare consideredtargetedarteries.[2]Intermsofmorbidityandmortality,atherosclerosisisamajorcause ofheart attacksandstrokesaswellasofmanyother cardiovascular conditions. Dueto itschronic nature and need for lifelong care, it causes over 17.9 million deaths annually, which has a significant impact on healthcare expenses.





ETIOLOGY:

1. 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.

2. DiabetesMellitus: Diabetesdamagestheactivityofendothelialcellsandraisesoxidativestress by causing insulin resistance and elevated blood glucose. These elements play a part in plaque development and artery irritation.

3. 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.

4. 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.

5. OxidativeStress: ResearchhasdemonstratedthatalargerratiobetweenROSandantioxidants mightexacerbatethedestructionofendothelialcells.Oxidativestressandirritation,twoimportant stages in the formation of plaque buildup, are triggered by ROS.

6. 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.

7. GeneticFactors: Atherosclerosis ispredisposedbyalterations ingenesrelatedto bloodvessel function, swelling, andthe breakdownoflipids. Inparticular, analterationinthe APOBgenecan alter how cholesterol is metabolized.

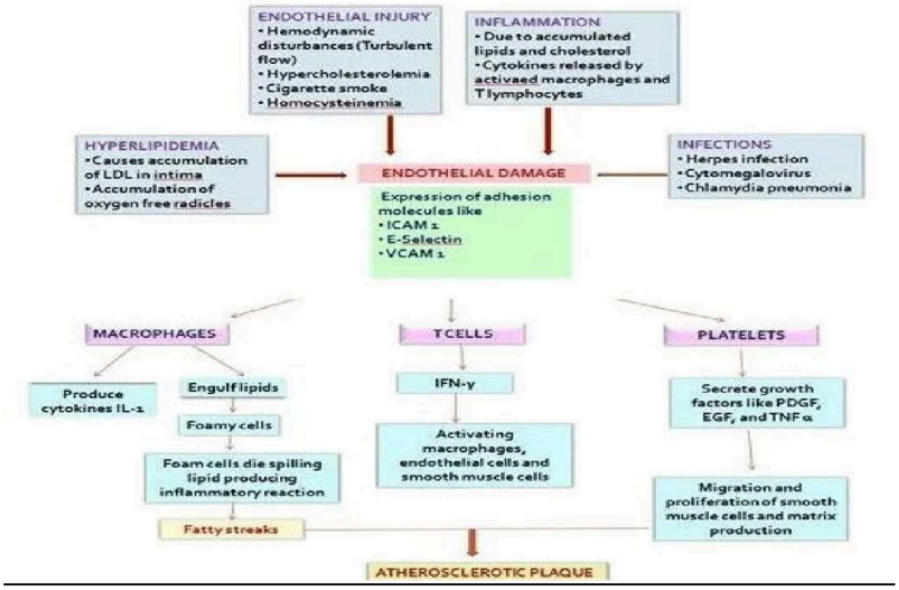
8. AgeandGender: Aspeopleage,theirarterywallsundergofunctionalandstructuralshiftsthat are marked by increased levels of oxidative stress and decreased flexibility. Women's risk increases after post menopause since theyhave declining estrogen concentrations, but men's risk increases throughout their youth owing to fluctuating hormones.

EPIDEMIOLOGY:

Cardiovascularillnessisoftenregardedas"thediseaseofthecentury"consideringit istheutmost prevalent cause of impairment and early death globally, with cardiovascular pathologies accountingfornearlyhalfofdeaths[13].Accordingtostudyfindings,422.7millionindividualshad 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 occurthere.Thisdelaysidentificationbeforetheclinicalperiod,increasingearlydeathfrom

CVD.[14]Conversely, wealthycountries haveseenarise intheprevalence and incidenceofCVD resulting from lack of exercise, high levels of alcohol and smoking, and an imbalanced diet.

PATHOGENESIS:



RISKFACTORS:

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 inflammatoryprocesses,hyperglycemiaandresistancetoinsulinpromoteatherosclerosis.Therisk isincreasedbyexcessweight,especiallyabdominalobesity,whichiscloselyassociatedwithlipid 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.

CLINICALPRESENTATIONS:

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.Thearteriesimplicatedandtheextentoftheblockageaffecttheclinicalmanifestations.

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 occasionallybefeltintheback,neck,jaw,shoulders,orarms.Usually,thediscomfortgetsgreater as you move and goes awaywhen you relax. The discomfort mayalso be triggered byemotional stress.

- Chestpain
- Nauseaandvomitingsensation
- Fatigue
- Weakness
- Shortness ofbreath
- Depressionandanxiety
- Heart attack
- Coronarythrombosis

DIAGNOSIS:

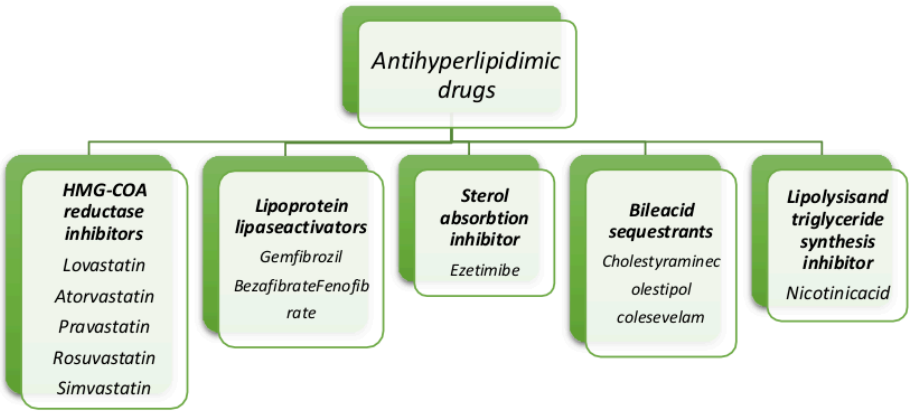
1. Clinical Assessment: To evaluate risk variables such as tobacco use, hypertension, diabetes, and family histories of coronary illnesses, an extensive medical history and physical assessment arecrucial.Atherosclerosisriskmaybeindicatedbydecreasedperipheralpulses,carotidbruits,or hypertension.

2. BloodTest:LipidProfile: HighLDL,total,andtriglyceride levelsand lowHDLcholesterol are significant markers of atherosclerosis.
ExtremelySensitiveHigh-sensitivityC-reactiveprotein(hs-CRP):Elevatedlevelsofhs-CRP indicate systemic inflammation and an increased likelihood of cardiovascular events.
FastingBloodGlucose:Elevatedbloodsugaroraberrant glucose metabolismare frequently linked to atherosclerosis.

3. ImagingTechniques: Ultrasound:Dopplerultrasonographyofthelowerlimbsorcarotid 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 producesextremelyhigh-resolutionimagesofbloodarteriesandcanidentifycoronaryheart disease or peripheral artery disease.

PHARMACOLOGICALMANAGEMENT:-

ANTI-HYPERLIPIDEMICAGENTS:



CLASSIFICATIONOFANTIHYPERLIPIDEMICDRUGS

HMG-COAREDUCTASE 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 inhibitedbystatins.Thislowersintracellularcholesterolcontentandincreasestheactivityof 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 totalcholesterolwhile slightly increasing HDL cholesterol.

Dose: Thedosevariesdependingonthespecificstatinandpatientfactors

- Atorvastatin :10–80mgoncedaily
- Rosuvastatin :5–40mgoncedaily
- Simvastatin :10–40mg oncedaily(maximum80mginspecificcases)
- Pravastatin :10–40mgoncedaily

LIPOPROTEINLIPASEACTIVATORS:

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.LPLactivatorsincreasetheactivityofLPL.TriglyceridesfoundinVLDLandchylomicrons arehydrolyzedbyLPLtoproduceglycerolandfreefattyacids.Tissuessubsequentlyabsorbthese unboundfattyacidsfordepositionorenergygeneration.Thisproceduremakesiteasiertoremove triglyceride-rich lipoproteins from the circulation and lowers the amount of triglycerides in the blood.

Dose:

- Gemfibrozil : 600mgtwicedaily, 30minutesbeforemeals.

STEROLABSORPTIONINHIBITORS:

Mechanism of Action: The Niemann-Pick C1-Like 1 protein that is present onthe brush border of the tiny intestine's wall is inhibited by ezetimibe. Its malfunction prevents the body from absorbingdietaryandbiliarycholesterol,whichresultsinfewerlipidsenteringtheliver.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:

- Theusualdoseofezetimibeis10mgoncedaily,withor withoutfood.

BILEACID 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:4–24gdaily,dividedinto1–6doses, mixedwithwateror juice.

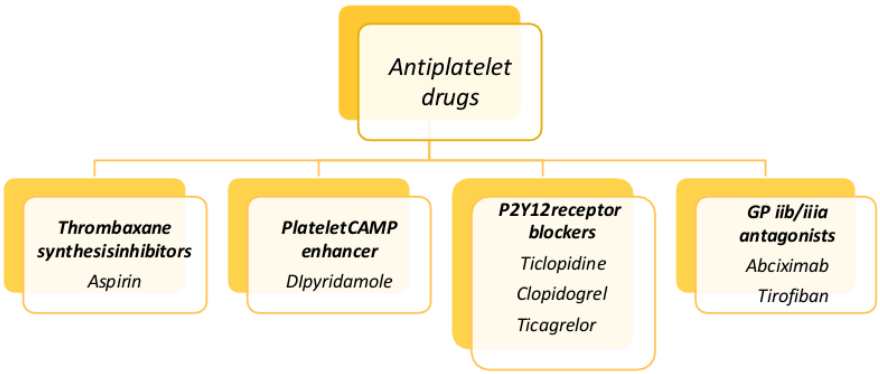
LIPOLYSISANDTRIGLYCERIDESYNTHESIS INHIBITORS:

Mechanismofaction: Nicotinicacidreducestheactionoflipolysis instoredfatbybindingtoG- protein-coupledreceptorsonfatcells,whichresultsinaninhibitionin thereleaseofunboundfatty 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:

- Initialdose: 250mg daily,takenwith food.
- Maintenancedose:Graduallytitratedto1.5–2g/day,dividedinto2–3doses.

ANTI-PLATELETAGENTS:



CLASSIFICATIONOFANTI-PLATELETDRUGS

THROMBOXANESYNTHESIS INHIBITORS:

Mechanism of action: Cyclooxygenase-1 in platelets is eternally inhibited by inhibitors of thromboxane A2 production, such as aspirin. Because ofthis inhibition, arachidonic acid cannot beconverted intothromboxane A2,apotent vasoconstrictorthat encouragesplatelet aggregation. This inhibition is irreversible by nature. For the full 7–10 day lifetime, platelets are unable to synthesize thromboxane A2.

Dose:

- Aspiriniswidelyutilizedforitsblood-thinningpropertiesatlowdoses(81–100mgdaily).
- Aloadingdoseof150–300mg,followedby75–100mgperday,isadvisedforacute coronary syndromes.

PLATELETCAMPENHANCERS:

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:

- Incombinationwithaspirin, take 75–100mg ofdipyridamole fourtimes a dayor 200mg twice a day.
- Cilostazol:100mgtwiceadayforvasodilatoryandantiplateleteffects.

P2Y12RECEPTOR BLOCKERS:

Mechanism of action: Clopidogrel, ticagrelor, and prasugrel are examples of P2Y12 inhibitors thatblocktheplatelets'P2Y12receptor.ThisreceptorisnecessaryfortheADP-inducedactivation 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:75mgeverydayafteraloading doseof300–600mg.
- Prasugrel:10mgper dayaftera60mgloadingdosage.
- Ticagrelor:90 mg twiceadayafteraloading doseof180mg.

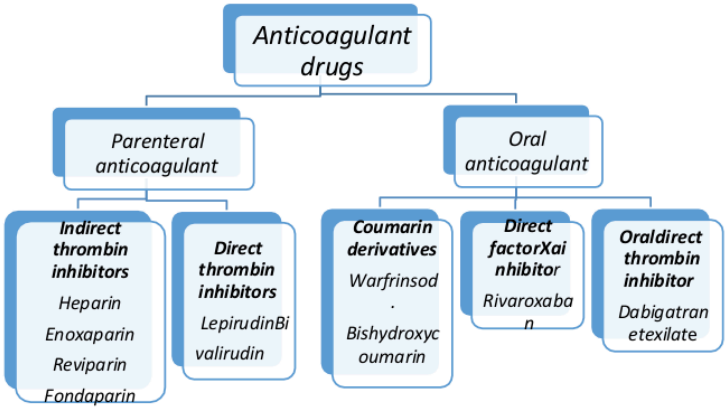
GLYCOPROTEINIIB/IIIAANTAGONISTS:

Mechanism of action: GPIIb/IIIa antagonists, such as tirofiban, eptifibatide, and abciximab, occupyplatelet GPIIb/IIIa receptors and serve as the main pathway for platelet clumping. These medicationspreventplateletaggregationbypreventingvonWillebrandfactorandfibrinogenfrom attaching to the receptor.

Dose:

- Abciximab:anIVbolusof0.25mg/kg,followedbya12-hourinfusionof0.125 mcg/kg/min.

ANTICOAGULANTS:



CLASSIFICATIONOFANTICOAGULANTDRUGS

INDIRECTTHROMBININHIBITORS:

Mechanism of action: Indirect thrombin inhibitors, namely low molecular weight heparins (LMWHs), including enoxaparin and unfractionated heparin (UFH), boost the function of antithrombinIII.Thisinteractionleads totheinactivationoffactorXaandthrombin,alsoknown

asfactorIIa.WhileUFHsuppressesboththrombinandfactorXaactivity,LMWHinhibitsa significant portion of factor Xa's activity due to its shorter molecular chain.

Dose:

Enoxaparin:

- 40mggivenviasubcutaneousinjectiononceperdayforprophylaxis.
- Subcutaneousdosage of1mg perkg every12hoursisthe treatment.

DIRECTTHROMBININHIBITORS:

Mechanismofaction: TheDTImedicationsareargatroban,bivalirudin,anddabigatran.DTI interacts primarilywith factor IIa, the thrombin sites of action. As a result, they inhibit fibrinogen's thrombin-dependent cleavage into fibrin. By blocking the connection with fibrinogenand thrombin, clotting becomes lessprevalent. Antithrombin III isnot involved in DTI's actions.

Dose:

- Dabigatran:Inatrialfibrillation,150mgtwicedailyisrecommendedforstrokeprevention (reduce to 75 mg twice daily in renal impairment).
- Bivalirudin:Aninfusionof1.75mg/kg/hourfollowinganinitialintravenousbolusof0.75 mg/kg for the purpose of percutaneous coronary intervention.

COUMARIN DERIVATIVES:

Mechanismofaction: VitaminKepoxidereductase,orVKORC1,isinhibitedbywarfarinaswell as coumarin derivatives. Thus, it decreases the process ofactive vitamin K and prevents clotting factorsII,VII,IX,andX,alongwithproteinsCandS,frombeingcarboxylated.Theanticoagulant impact of active clotting factors is delayed because the bodytakes longer to deplete them.

Dose: Forthefirst1-2days,theinitialdosageis5-10mgtakenorallyonceday,itisthenincreased to maintain an INR of 2-3 (for the majority of reasons).

DIRECTFACTORXAINHIBITORS:

Mechanismofaction: ProthrombinproductionispreventedbydirectfactorXainhibitorssuchas 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 dayfor 21days, followed by20 mg once daily for pulmonary embolism (PE) and deep vein thrombosis (DVT).

- Apixaban:5 mgtwicedaily(downto 2.5mgtwicedailyundercertaincircumstances) is the advised dosage for atrial fibrillation stroke prevention.

NONPHARMACOLOGICALMANAGEMENT:

1. NUTRITIONALMEASURES:

Consuming red meat and full-fat dairy products includes saturated fats, which increase LDL cholesterollevels.Healthyfats, amongthose innuts,fish,andoliveoil, maybeutilizedto reduce cholesterollevels.Consumingexcessiveamountsofsalt leadstohighbloodpressure,whichisthe largest risk factor for atherosclerosis. Cutting less salt intake can help lower blood vessel strain and regulate blood pressure.

2. PHYSICALACTIVITY:
Walking, cycling, and swimming are examples ofaerobic exercises that canraise HDL, or good, cholesterolwhileloweringLDLandtriglyceridelevels.Byreducingboththediastolicandsystolic 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.

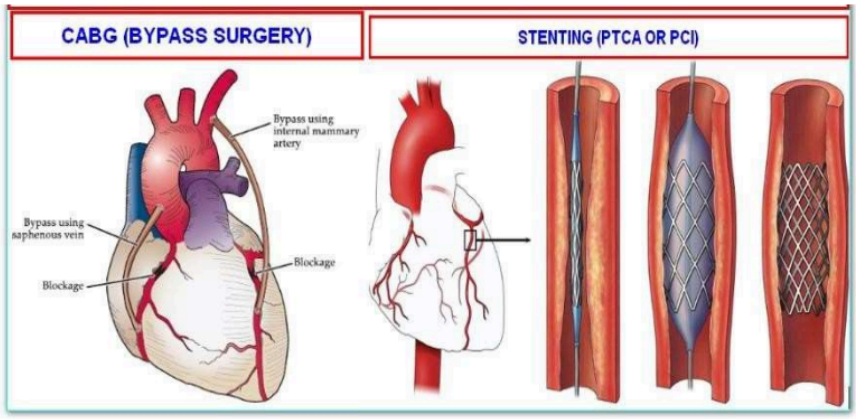
3. SMOKING QUITTING:
When quitting smoking, endothelial function enhances vasodilation and overall circulatory function. Smoking constitutes among the most significant underlying causes of cardiovascular disease.Givingupthisdangerousbehaviourreducestheriskofcardiovascularincidents,including coronary heart disease and stroke that are linked to atherosclerosis.

SURGICALMANAGEMENT:

1. CORONARYARTERYBYPASSGRAFTING,ORCABG:
CABG is a formofsurgerythat employs grafting fromvarious bodyareas, including the internal mammaryarteryor saphenousvein, to bypassblocked coronaryarteries. Byproviding adifferent path for the flow of blood, CABG greatly enhances the supply of oxygen that gets to the heart muscle. Patientswithdiabetes, left main heart disease, ormulti-vesseldisease canbenefit greatly fromit. Major coronaryarterydisease canbe effectivelytreatedwiththis operationover the long term, which lowers symptoms and increases survival.

2. PERCUTANEOUSTRANSLUMINALCORONARYANGIOPLASTY(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 arteryblockages and is frequently performed in conjunction with stent insertion. It is perfect for individuals who may not be able to handle openheart surgeryand has a quicker recoveryperiod.

3. INTRA-AORTICBALLOONPUMP(IABP):
Whenseverecardiacfailure,includingcardiogenicshock,isoccurringinapatient,theIntra-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" therapyapproach.



CABGVS.STENTING:CORONARYARTERYTREATMENTOPTIONS

METHODOLOGYMATERIAL

SANDMETHODS

STUDYDESIGN	PROSPECTIVEOBSERVATIONALSTUDY
SAMPLESIZE	100
STUDY SITE	MULTI SPEACIALITY HOSPITAL
DEPARTMENT	CARDIOLOGY
STUDY DURATION	6 MONTHS

SOURCEOFDATA AND MATERIALS:

- Patientsconsentforms
- Labreports/angiogramreport

- Patientdatacollection form
- Patientsprescription

INCLUSIONCRITERIA:

- Ageabove18years.
- Patientswithcardiovasculardisease/ atherosclerosis
- Patientswhoarewillingtoparticipateinthe study

EXCLUSIONCRITERIA:

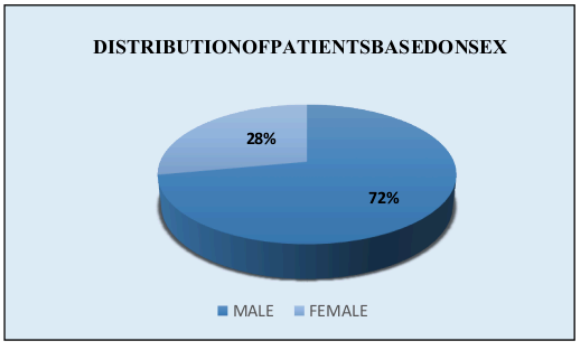
- Patientswith impairments
- Pregnantwomen,lactating
- Patientsbelowthe ageof18

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 extentofarterialstenosis.Patientswerethenmanagedaccordingtostandardtherapeuticstrategies, including pharmacological treatments such as statins, antiplatelets, antihypertensives, and antidiabetic agents, along with non-pharmacological approaches like lifestyle modifications.

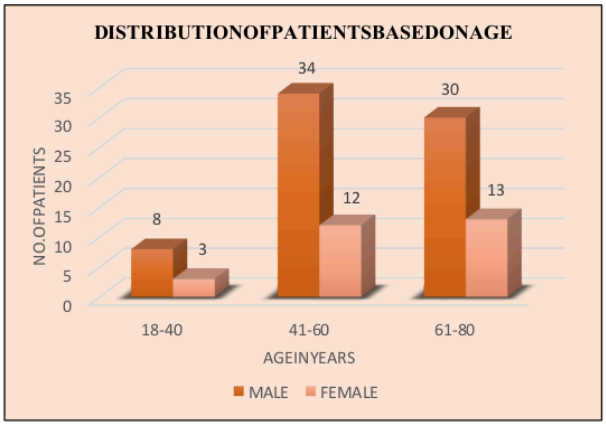
RESULTS

1. DISTRIBUTIONOFPATIENTSBASEDONSEX



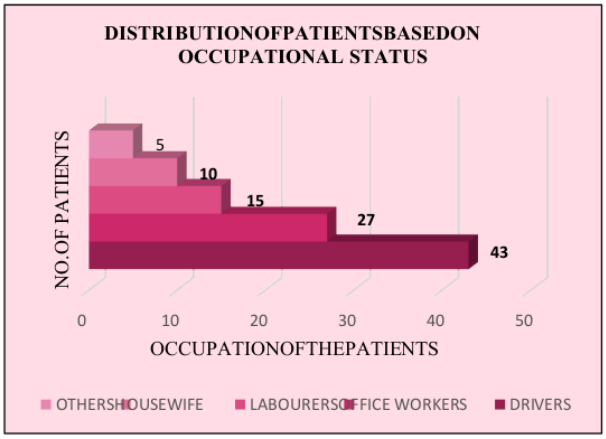
Here itwasobservedthatbasedonsex72patientsweremaleand28patientswere female.

2. DISTRIBUTIONOFPATIENTSBASEDONAGE



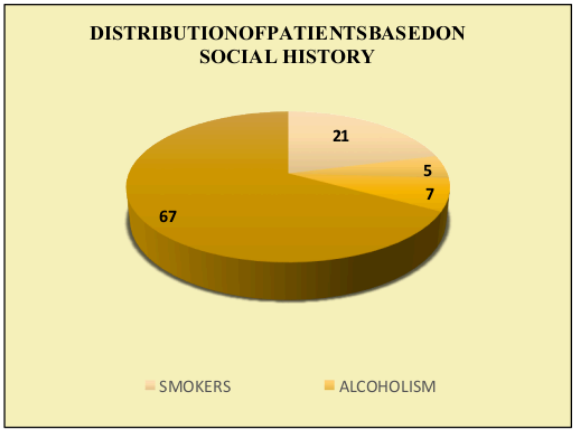
Atherosclerosis in the age of 18-40 years males were found to be 8 and females were 3, 41-60 years maleswere34and femalewere12andattheageof61-80maleswere30and femaleswere 13.

3. DISTRIBUTIONOFPATIENTSBASEDONOCCUPATIONALSTATUS



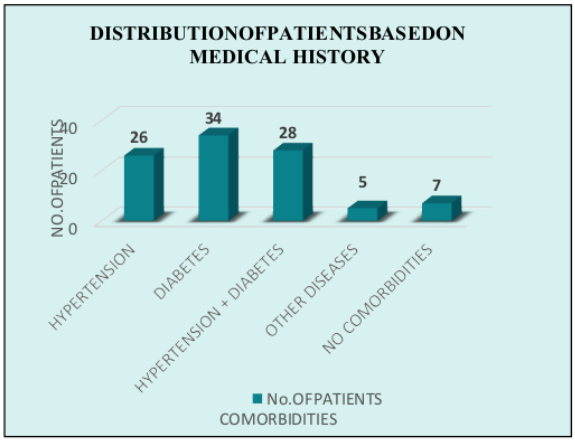
Here we observed that distributionofdisease inthe patients based ontheir occupationalstatus in whichdriverswere found43, 27wereofficeworkers, labourerswere15, 10werehousewifeand 5 were others.

4. DISTRIBUTIONOFPATIENTSBASEDONSOCIALHISTORY



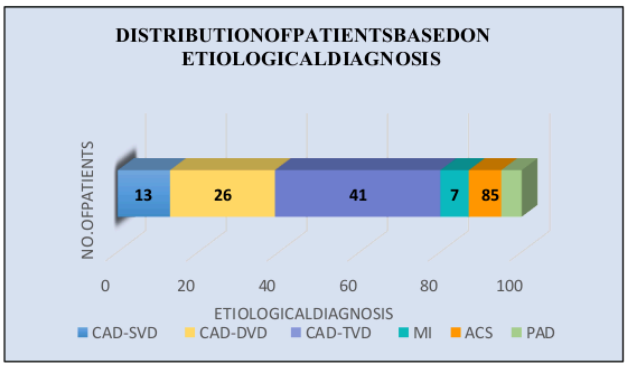
Patients with the social history i.e., smokers were found to be 21, 5 patients with alcoholism, 7patients were tobacco chewers and 67 patients were with no social history.

5. DISTRIBUTIONOFPATIENTSBASEDONMEDICALHISTORY



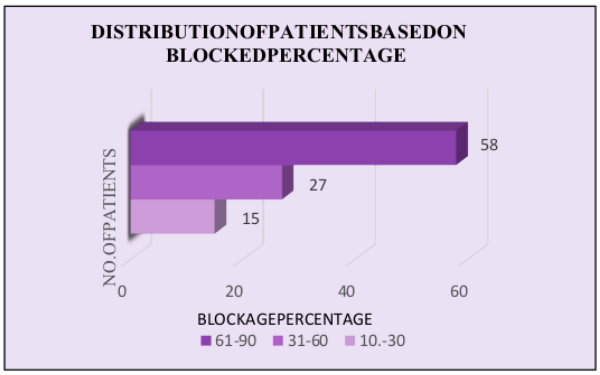
Patients with comorbid conditions such as Hypertension were found to be in 26 patients, 34 patientswerediabetic,28patientswerefoundincombinecomorbidconditioni.e.,Hypertension +Diabetes,5patientswithother diseasesand7patientswerewithnocomorbid conditions.

6. DISTRIBUTIONOFPATIENTSBASEDONETIOLOGICALDIAGNOSIS



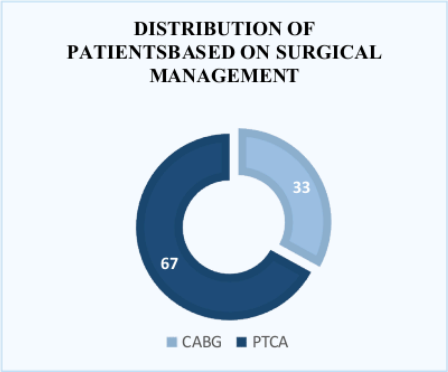
CAD-SVDwasdiagnosedin13patients,26patientswerediagnosedwithCAD-DVD,41patients were diagnosed with CAD-TVD, 7 patients diagnosed with MI, 8 patients diagnosed with ACS and 5 patients were diagnosed with PAD.

7. DISTRIBUTIONOFPATIENTSBASEDONBLOCKEDPERCENTAGE



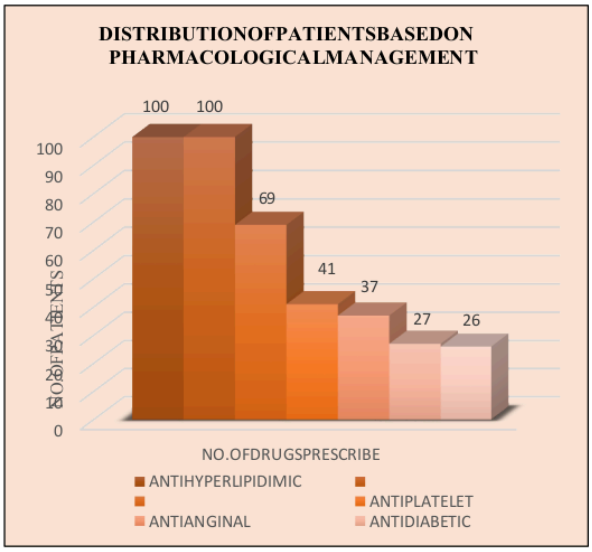
Hereweobservethat10-30%ofblockagewasfoundin15patients,27patientshad31-60% blockage, 61-90% of blockage was found in 58 patients.

8. DISTRIBUTIONOFPATIENTSBASEDONSURGICALMANAGEMENT

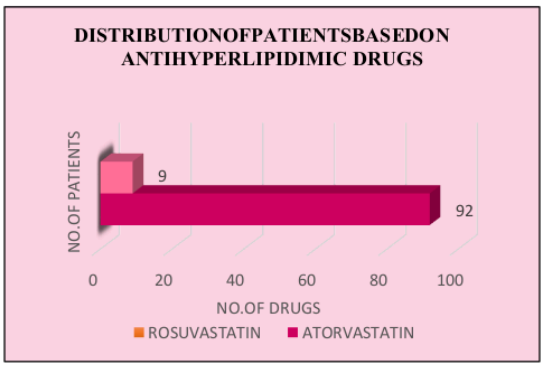


Here we observe that 33 patients were undergone for CABG and 67 patients were suggested for PTCA surgical management.

9. DISTRIBUTIONOFPATIENTSBASEDONPHARMACOLOGICALMANAGEMENT

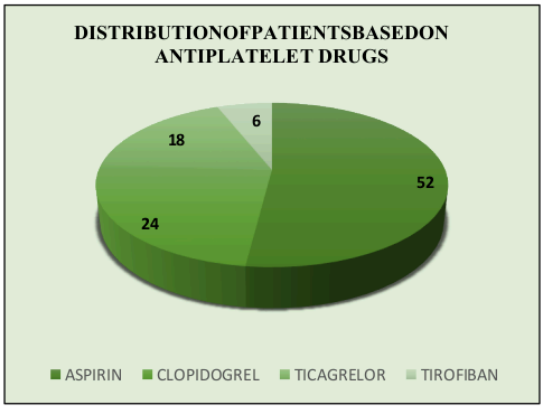


10. DISTRIBUTIONOFPATIENTSBASEDONANTIHYPERLIPIDIMICDRUGS



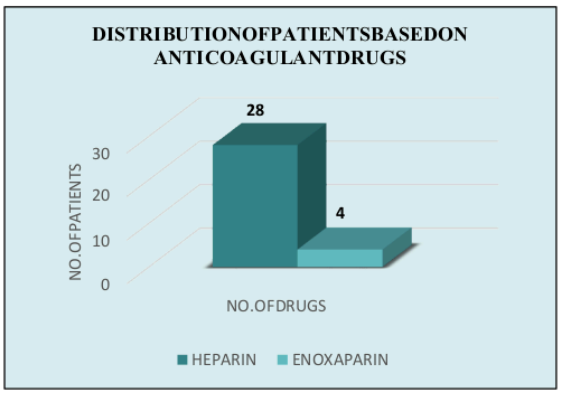
Atorvastatinwasprescribedin92 patients;8 patientswereprescribed Rosuvastatin.

11. DISTRIBUTIONOFPATIENTSBASEDONANTIPLATELETDRUGS



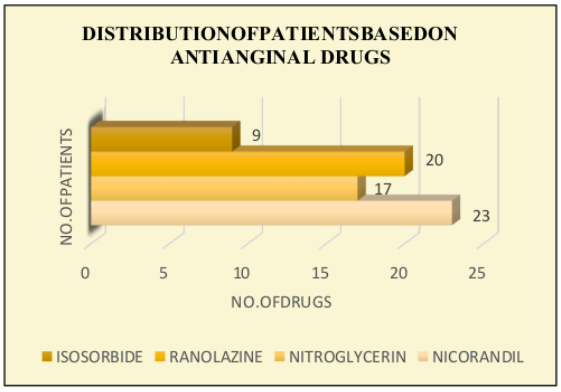
Aspirin was prescribed in 52 patients, 24 patients were prescribed clopidogrel, 18 patients were prescribed ticagrelor and tirofiban was prescribed in 6 patients.

12. DISTRIBUTIONOFPATIENTSBASEDONANTICOAGULANTDRUGS



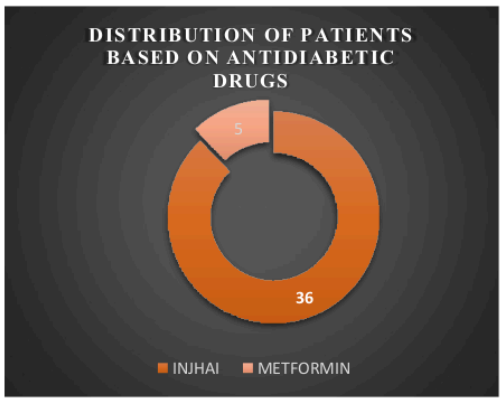
Itwasobservedthatheparinwasprescribedin28patientsand4patientswereprescribedenoxaparin.

13. DISTRIBUTIONOFPATIENTSBASEDONANTIANGINALDRUGS



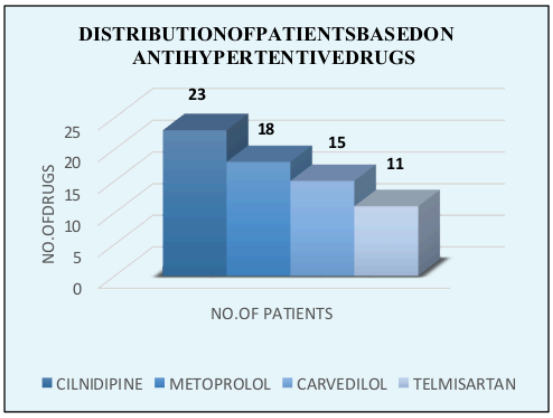
Nicorandilwasprescribedin23patients,17patientswereprescribednitroglycerin,20were prescribed ranolazine, and isosorbide was prescribed in 9 patients.

14. DISTRIBUTIONOFPATIENTSBASEDONANTIDIABETICDRUGS



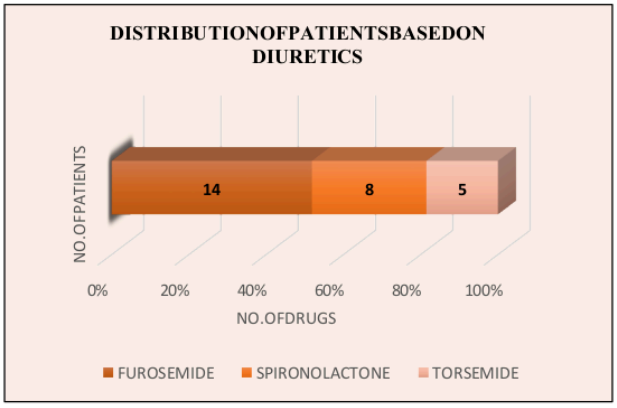
Inj.HAI wasgivenin36patients,5patientsweregivenmetformin.

15. DISTRIBUTIONOFPATIENTSBASEDONANTIHYPERTENTIVEDRUGS



Cilnidipine was prescribed to23 patients,18 patients were given metoprolol,15 patients weregiven carvedilol, telmisartan was prescribed in 11 patients.

16. DISTRIBUTIONOFPATIENTSBASEDONDIURETICS



Furosemidewasgivenin14patients,8patientsweregivenspironolactone,torsemidewas prescribed in 5 patients.

DISCUSSION

Inourstudy,weobservedthat antihyperlipidemicsandantiplatelets werecommonlyprescribed for thetherapeutic management ofatherosclerosis andother relateddisorders. Furthermore,the majorityof the patients included in our studywere 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

Themostcommonsurgicalprocedureinour studyisPTCAfollowedbyCABG

Themostcommonlygivenantiplatelet isaspirin(brandnameEcospirin),followedbyclopidogrel (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.

REQUIREMENTSFORFUTUREDRUGDEVELOPMENT:

1. ADDRESSINGRESIDUALRISK:
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 geneticallydetermined, hereditary lipoprotein linked to cardiovascular risk,isanotherpossibilitythat isgaininginterest.TreatmentsthattargetRNA-basedinhibitorsare being researched to lower levels of lipoprotein.

2. ENHANCEDSAFETYPROFILES:
Safetyshouldcomefirstwhiledevelopingtreatmentsforatherosclerosis.Infact,eventhoughthere isn't anyconcrete proofofmuscle injury, a significant number ofpatients 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

3. COST-EFFECTIVENESSANDACCESSIBILITY:
BecauseoftheirhistoricallyhighprICes,modernmedicineslikePCSK9inhibitorsandmonoclonal antibodiesare notaswidelyavailableworldwide. Thecreationofbiosimilarsandsmall-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 structuresthatguaranteereasonablypricedpharmaceuticals.Fairnessandequityinaccesswillalso be greatly aided by grassroots education and initiatives to upgrade the healthcare system in low- and middle-income (LMIC) nations.

4. LONG-TERMEFFICACYSTUDIES:

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 recordsshouldguaranteethegatheringofempiricaldataafterapproval. Thisapproachwillensure 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 managementofatherosclerosiswithdifferentclassofdrugs.Thetherapeuticstrategiesincludethe useofantihyperlipidmicagentssuchasatorvastatinwhichreduceslowdensitylipoprotein(LDL). Antiplatelet medicationsareusedtoprevent thestickingofplateletstogetherandformingclumps 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 followedbymyocardialinfractionandacutecoronarysyndrome.Thepatientswithcoronaryartery disease was treated with aspirin and clopidogrel and patients with acute coronary syndrome was treated withstatinand antiplatelet therapies. Antianginaldrugs suchas ranolazine and nicorandil areprescribedbasedonsymptoms.Optimizingtreatmentthroughpersonalizedtherapyandregular 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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APROSPECTIVEOBSERVATIONALSTUDYONTHEEVALUATION OF THERAPEUTIC STRATEGIES IN THE MANAGEMENT OF ATHEROSCLEROSIS: A COMPHREHENSIVE CLINICAL AND PHARMACOLOGICAL APPROACH

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