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RESEARCH ARTICLE

ULTRASOUND AND DYNAMIC CONTRAST CT IN THE DIAGNOSIS AND DETERMINATION OF THE CLINICO-IMAGING OUTCOME IN ACUTE PORTAL VEIN THROMBOSIS

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Abstract

Background: Acuteportalveinthrombosis(APVT)hasdevastatingconsequences,ifnotidentifiedearly.However,thenon-specificpresentation rendersclinicaldiagnosisdifficult.Theroleof imaginginthisdiseaseis,therefore,crucial.Thisstudyaimstoevaluatetheroleofcolor Doppler anddynamiccontrastCTintheearlydiagnosisofAPVTandtodeterminethecl inico-imagingoutcomeofthesepatients.
Methods: FortypatientsdiagnosedwithAPVTfrom2019-2022atthe DepartmentofRadio-diagnosisandImagingwereputonappropriatetherapybythetreating physiciansandwerefollowedupforradiologicaloutcome.
Results:APVTwasdetectedoncolor Dopplerin92.5%of thepatientsintheformofechogeniccontentswithinPortalVein(PV)and complete/partialabsenceofcolorflow.Intermsofpatients,42.5%hadadilate dPV.Thirteen(32.5%)patientsshowedsuccessfulre-canalizationofthethrombosedportion,whiletwenty-one(52.5%)patientsadvancedtocavernomatoustransformationofthePV.Pri ortotheinitialfollow-up,six(15%)patientspassedaway.
Conclusion: DuplexDopplerisaneffectivescreeningmethodfor APVT.CECTshouldbeutilizedforconfirmation,though.Thepatientswhore ceiveearly anticoagulationhave amarginallysuperiorAPVToutcome.

Introduction:-

PortalVeinobstructionreducesbloodflowtotheliverbyasmuchastwo-thirds.ThePVdoesnot haveanauto-regulatorymechanismofitsown.Theadenosinefailuretheory[1-4],whichproposes thatadenosine,avasodilator,isproducedintotheinterstitiumthatencloseshehepaticarterioles andportalvenules,statesthatbloodflowtotheliveris,thankfully,preserved.Inahealthystate, portal bloodflowflushesoffregionaladenosineconcentrationsfromthesiteofthearterial resistance.Lessadenosineiswashedawayandthelocalconcentrationofadenosinerisesifthe portalflowisreduced.Hepaticarterialdilatationistheeffectofthis.Asaresult,theso-called“hepaticarterialbufferresponse”[5]occurswhentheportalflowisreduced,whichcausesthe hepaticchanneltodilate,andviceversa.Thesituationchangeswhen thethrombosisisrestrictedto oneoftheportalbranches,whichcausestheafflictedlobetoatrophyandthecontralaterallobeto enlarge[6].

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Onallimagingapproaches, Acute PortalVeinThrombosisisfrequentlyassociatedwithincreasedhepaticarterialbloodflow[7,8].Because of glycogen depletion, fat deposition in specific areas, or decreased contrast material delivery, the hepatic segment fed by an occluded portal venous branch may look comparatively hypodense. However, on dynamic scans acquired right after a bolus IV injection of contrast material, the attenuation of this segment may paradoxically increase due to compensatory enhancement of the local hepatic arterial flow.In the lumen of the portal vein, PVT is typically visible on CT images as a non-enhancing filling defect. The clot is typically isodense or slightly hypodense in comparison to the nearby soft tissues. On contrast-enhanced images, thrombus that has just developed (less than one month old) may be hyperdense and obscured. In these contraststudies, researchhasshownthatimagingcorrelatesfor PVTincludedilatedbiliarychannelsortumorcompressionof theportalvein[9].WhenPVTissuspected,ultrasoundisthefirstlineofimagingwithanaccuracyrangingfrom88-98%andsensitivityandspecificityof80-100%innearlyallofthestudies[10-12].A thrombus is seen as a hypo/isoechoic material filling part of (partial thrombosis) or the full vessel on Gray-Scale ultrasonography (complete thrombosis). A condition known as "cavernomatous transformation" or "cavernoma," which can also be readily identified with Doppler-ultrasound, can ultimately cause the normal portal vein to be replaced by numerous tortuous vessels with hepatopetal flow. To distinguish between high degree partial thrombosis and full thrombosis, it is mandatory to use color/power and pulsed Doppler to determine whether the vessel has a remnant blood flow.

Therearedrawbacksto ultrasonography,suchasanabundanceofintestinalgasandtheinabilitytodetectmesentericischemia.However,inpatientswithagoodacousticwindow,ultrasoundissufficienttodetectPVT[Fig.1].Asecond-linecross-sectionalimagingtechniqueisusedtodiagnosethepresenceor absenceofPVTwhenultrasonographyisinsufficient.Contrast-enhanced4Phase(pre-contrast, arterial,portal,andlate)CT(CECT)andcontrast-enhancedMRI(CEMRI)arebothoptionsthat canbeused,withCTbeingadvisedinunstableindividualswhohaveacuteabdominalsymptoms[Fig.2].Diagnosisofmesentericischemia,septicfoci,intra-abdominalmalignanciesandbetter sensitivityintheidentificationofthrombosisinthesplenicandsuperiormesentericveinsare amongthebenefitsofMRandCToverultrasound.Althoughitisnotcurrentlyrecommendedfor useinclinicalsettings,unenhancedmagneticresonanceportographyisbeingexplored[13-15].

OncePVT hasbeen detectedsonographically,CECT orCEMRIareneededtodeterminethe degree ofthrombosisandtomakeitpossibletomapoutalloftheporto-systemiccollaterals, bothofwhich arecrucialforthedevelopmentofre-canalizationtherapy.

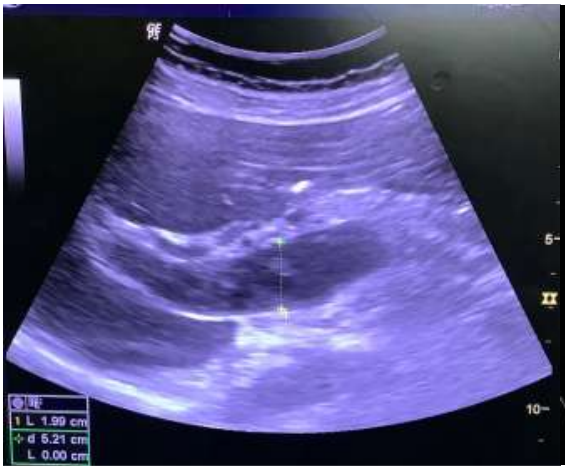

Fig1:- Gray-scaleultrasoundpictureshowing distendedPVwithheterogenouscontents withinitslumen.

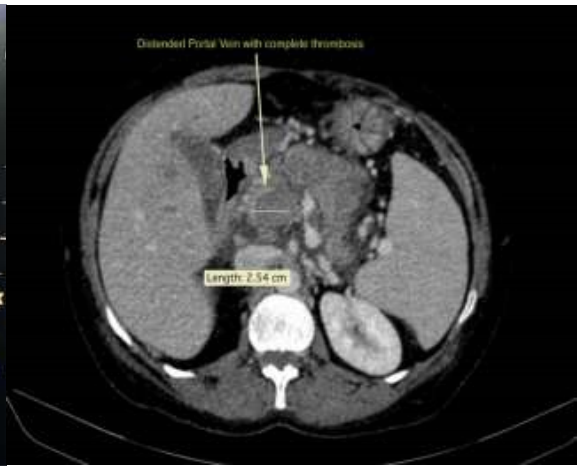

Fig 2:- Axial CECTin the portal phase showing complete thrombosis of Portal Vein lumen.

Recanalization-focusedtherapiesmayhaveanegativeimpactonclinicaloutcomesinAPVTpatientsbecauseofthedelicatebalancebetweenthrombosisandbleedinginthesepatients.For manyyears,oralvitaminKantagonistshavebeenthego-

totreatmentoptionaftercommencing withun-fractionatedheparin[16]orlowmolecularweightheparin(LMWH)andthenbridgingto oralvitaminKantagonistsforlong-termanticoagulation[17].Accordingtothefindingsofarecent investigation,sametreatmentapproachesshouldbeusedbecausetheprognosisofincidentally identifiedsplanchnicveinthrombosisissimilartothatofclinicallydiagnosedsplanchnicvein thrombosis[18].Currentrecommendationsforearlyanticoagulationinpatientswithacute PVTare, however,basedonlimitedevidence.Therehavebeenreportsintheliterature[19-21]ofthrombolytictherapyforacutePVT administeredbytranshepatic,transjugularroutes andretrogradelythroughomentalveins.The portalveinhas beensuccessfullyrecanalizedusingtransplenicaccessintotheportalsystemfortheinstallationof portosystemicshunts[22,23].However,onlyasmallnumberofformaltrialshavebeenconductedtoestablishitsspecificsignifican cein clinicalcontexts[24-26].

Aimsandobjectives:-

Thestudysoughttoinvestigatetheeffectivenessofcolor Dopplerand dynamiccontrastCTinthe identificationofacuteportalveinthrombosisaswellastheclinicalandradiologicaloutcomesof patientswithacuteportalveinthrombosis.

EthicalClearance

TheSher-i-KashmirInstituteofMedicalSciences(SKIMS),Srinagar,InstitutionalEthicsCommitteewas consultedbeforethestudybeganinordertoobtain ethicalapproval.

Materialsandmethods:-

ThestudywascarriedoutintheDepartmentofRadio-diagnosisandImaging,SKIMSSrinagarin collaborationwiththeDepartmentsofMedicalGastroenterologyandSurgicalGastroenterology.BetweenJuly2019andJanu ary2022,fortypatientswhowereadmittedwithAcutePortalVein Thrombosis to thehospitalwerestudied.Allpatientsgavetheirapprovaltoparticipatein thestudy. UltrasoundPorto-splenicduplexscanningwasperformedusingGELOGIQP5,withstandard presets.Curvilinearprobeswithfrequenciesof5-7MHzandstandardsettingswereusedtoassess thePorto-splenicaxisaftermorethaneighthoursoffasting.Real-timesonographywasusedto locatetheportalveininsupineandlateraldecubituspositions.Thepresenceofdistendedsplenic, portalormesentericveinsfilledwithhypooriso-echoicmaterialwasconsideredsuspiciousfor APVTongrey-scaleimaging.These patientsthen underwentcolorDopplerexaminationfocussing onthespleno-portomesentericaxis.Whenflowcouldnotbeshownevenaftercarefulmanipulation ofscanningparameters,aduplexDopplerexaminationoftheportalveinwasperformed.The completeabsenceofcolorflowandspectralwaveformsorthepresenceof remnantflowhelped categorizethepatientsintothosewithcompleteorpartialthrombosis.Forfurtherelucidation,the patients underwentsecond-lineimagingevaluationby wayofmultiphasedynamicCECTtoconfirmthepresenceofAPVT.TheCECTwasdoneon64-sliceCT:SOMATOMSensation(SiemensGermany).Non-ioniccontrast material(150ml,300mgI/ml) wasinjectedintravenouslyas abolushavingaflowrateof3.5ml/swithan18Gcatheterinserted inthebrachialvein. Multiphase CTimageswerethenevaluatedfordeterminingtheacutenatureofthethrombus(characterizedby theabsenceofcollateralveins);thediameterofthespleno-portalvenousaxis;theproximaland distalextentofthethrombus;thepresenceorabsenceofresidualflow;thepresenceofascites; bowelwallenhancementpattern;hepaticmorphologyandanyotherassociatedimagingfindings. Thepatientshavingchronicthrombosis(characterizedbythepresenceofcollateralveins)wereexcludedfromthestudy atthisstage.

Patientswhoreceivedanticoagulationwithin30daysofthediagnosiswerereferredtoasreceiving earlyanticoagulation,whiletheremainingpatientswhodidnotreceiveanticoagulationatall,or receivedanticoagulationafterthe30days-markwerecategorizedasreceivingnoearlyanticoagulation.Allthepatientswerereassessedby ColorDopplerordynamicmul tiphaseCECT asclinicallywarranted,toassessthepost-treatmentmorphologicalstatus.TheacutephaseCTscan wascomparedtothemostrecentCTscantakenduringthemonitoringphase(typicallytwoto threemonths,maximumoneyear,median90days),andpatientoutcomesweredividedintothree categories:recanalization;progressiontocavernomatoustransformationoftheportalvein(CTPV);anddeath.

Data Analysis:
The recorded datawere compiled and entered into a spreadsheet(Microsoft Excel)andthen exportedto the data editorof SPSS Version 20.0 (SPSS Inc., Chicago, Illinois, USA).Continuous variables were expressed as Mean ±SD and categorical variables were summarized as frequencies and percentages.Graphically, the data was presented by barand pie diagrams.The chi-square test or Fisher 's exact test, whichever was appropriate, was employed for the comparison of categorical variables. Ap-value of less than 0.05 was considered statisticallysignificant.All p-valueswere two-tailed.

Results:-

AcutePVTwasdetectedonColorDopplerin92.5%ofthepatients.Theultrasoundfindings consistedofechogeniccontentsintheportalveinandcompleteorpartialabsenceofcolorflow[Figs3and4].Intheremaining7.5 %ofpatients,DuplexDopplershowednormalcolorflowinthe mainportalveinbutthesepatientshadpartialbranchportalveinthrombosisondynamicCECT [Table1].

Table 1:- Detectionofacuteportalveinthrombosis on colorDoppler.

Acuteportalveinthrombosis	Number	Percentage
Present	37	92.5
Absent	3	7.5
Total	40	100

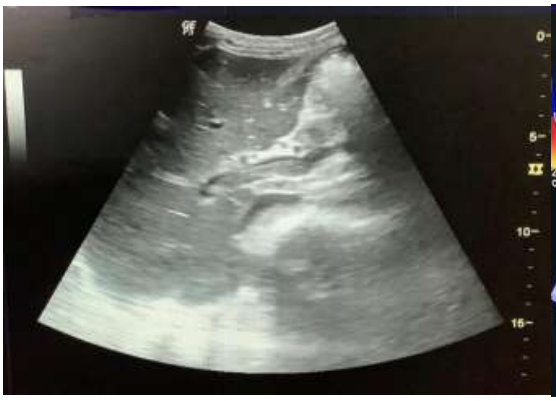

Fig 3:- Gray-scalesonogram showing echogenic contentswithinthemainportalvein.

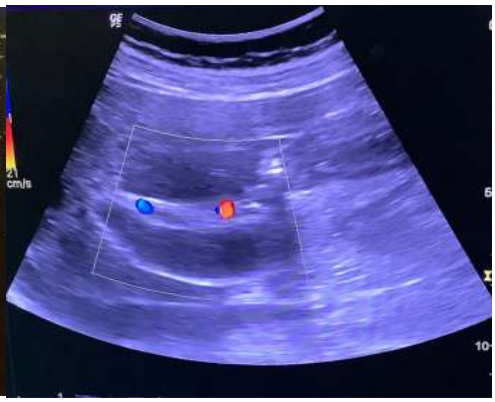

Fig4:- ColorDopplerimageshowingno detectableflowintheportalveintrunk.

Outofthestudypatients,42.5%hadaportalveindiametergreaterthan15mm.Thrombosisinvolvingthetotallumenwasseenin14patients(35%)&partialin26patients(65%)[Fig. 5].Thepercentagesofpatientshaving type2a,type1,type3andtype2bwere37.5%,30%,17.5%and15%respectively.Thesuperiormesentericveinwasinvolvedin8patients(20%)andthesplenicveinwasinvolvedin9patients(22.5%).Bowelwallenhancementwasnormalin90%ofthepatients whiletheremaining10%ofpatients(n=4)showedbowelwallabnormalitieslikegutwall thickening,hypo-enhancinggutwallormucosalhyper-enhancement.TransientHepaticAttenuationDifferences(THAD)wereseenin11ofthe25non-cirrhoticpatients(44%).THAD werediffusewiththecentral-peripheralphenomenonin8patients[Fig6]andsectorialwedge-shapedin3patients.Asciteswasdetectedin47.5%ofpatientsonCECT(n=19).Outofthe non-cirrhoticpatients,asciteswasdetectedin11 patients(44%) [Table 2].

Table2:- CECTfindingsofstudyparticipants atthetime ofdiagnosis.

Parameter	Findings	Number	Percentage
Diameteroftheportal vein	≤15mm	23	52.5
	>15mm	17	42.5
Thrombosis	Partial	26	65.0
	Complete	14	35.0
Extentofthrombosis	1	12	30.0

	2a	15	37.5
	2b	6	15.0
	3	7	17.5
SMVinvolvement	Yes	8	20.0
	No	32	80.0
Splenicvein involvement	Yes	9	22.5
	No	31	77.5
Bowelenhancement	Normal	36	90.0
	Abnormal	4	10.0
Hepaticchanges(innon-cirrhotics)	Yes	11	44.0
	No	14	56.0
TypeofTHAD	Diffusewithcentral/ peripheral	8	73.0
	Sectorialwedge-shaped	3	27.0
Radiologically detectable ascites	Yes	19	47.5
	No	21	52.5

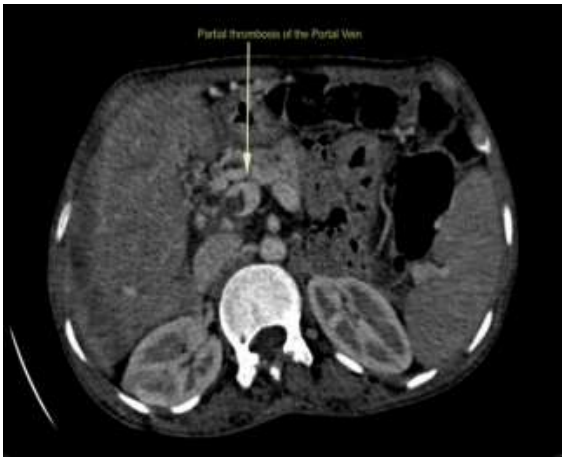

Fig5:- Axial CECTimageintheportalphase showingpartialthrombosisoftheportalvein.

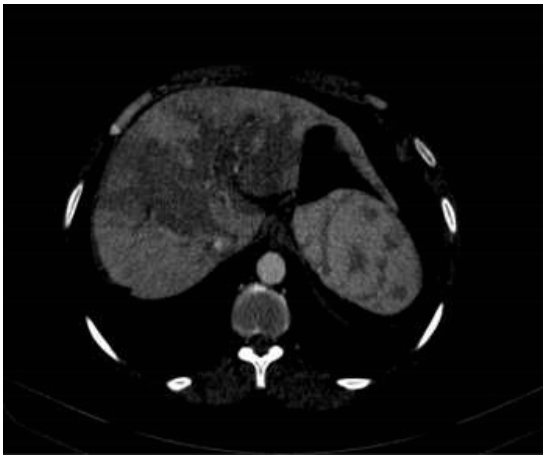

Fig6:-AxialCECTimageinthe arterialphase showing THADwiththecentral-peripheral phenomenoninapatientofAPVT.

Thirtypatientsreceivedearlyanticoagulation(within30daysofthedateofdiagnosis)accordingto therecentguidelineswiththechoiceofanticoagulantanddurationoftreatmentbeing individualizedbytheclinicianbasedontheriskofbleeding.Tenpatientscouldnotreceiveearly anticoagulationaspertheclinician'sdecision.AllthepatientswerethenfollowedwithColor Doppler or CECT as clinically warranted (usually 2-3 months, maximum 1 year)[Table 3].Doppler or CECT as clinically warranted (usually 2-3months, maximum 1year).

Table 3:- Earlytreatmentwith anticoagulants.

Early Treatment	Number	Percentage
Received	30	75.0
Notreceived	10	25.0
Total	40	100

Of thetotalpatients,52.5%progressedtotheCavernomatousTransformationofthePortalVein[Fig6].Re-canalizationofthethrombosedpartwasachievedin32.5%ofpatientsand15%ofpatientsdiedbeforethefirstfollow-upDoppler/CT [Table 4].

Table4:- Outcome ofstudyparticipants(Median90daysfollow-up).

Outcome	Number	Percentage
Re-canalization	13	32.5
Progressionto CTPV	21	52.5
Death	6	15
Total	40	100

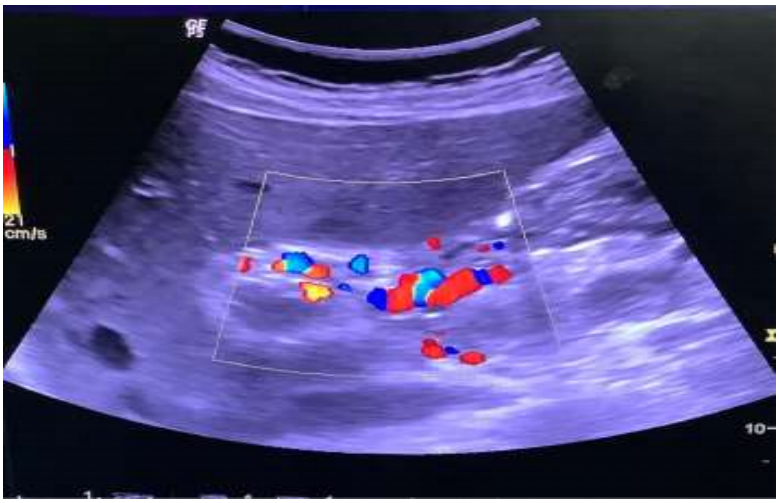

Fig6:- ColorDopplerimageshowing CavernomatousTransformationofthePortalVein.

Table 5:- Correlationofoutcomewith earlytreatmentamongstudysubjects.

Outcome	Earlytreatment received		Noearlytreatmentreceived		p-value
	Number	%age	Number	%age	0.121
Re-canalization	12	40	1	10	
Progressionto CTPV	15	50	6	60	
Death	3	10	3	30	
Total	30	100	10	100	

Outofthepatientstreatedwithearlyanticoagulation,re-canalizationwasachievedin40%ofthepatients;50%progressedtoCTPVand10%died.Onthecontrary,spontaneousre-canalizationwasseeninonlyoneoftheuntreatedpatients(10%);60%progressedtoCTPVand30%died[Fig7]. Thedifferenceinthe ratesofre-canalizationbetweenthetwogroupswas,however,notstatisticallysignificant.

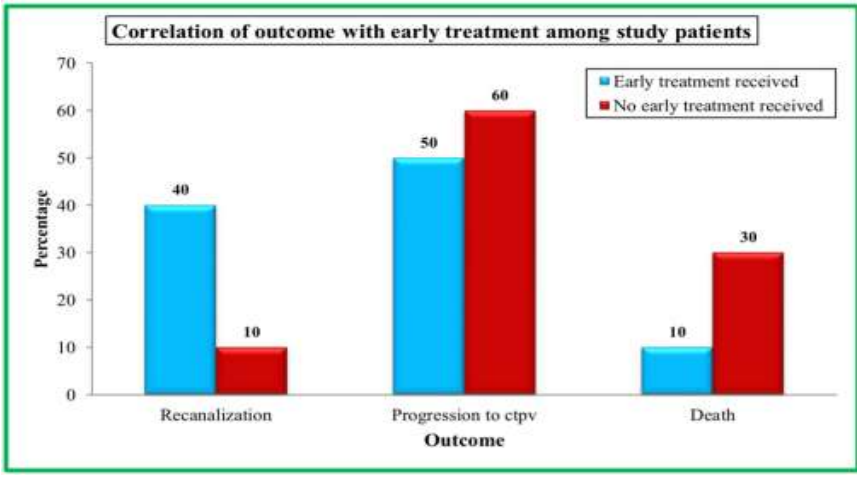

Fig 7:- Multiplebardiagramshowingthecomparisonofoutcomesinpatients receiving andnotreceivingearlyanticoagulation.

Discussion:-

ColorDopplerhasbeendemonstratedtobe89%sensitiveindetectingportalveinthrombosis,accordingtoresearchbyTessler FN etal[10].Otherstudieshaddemonstratedthat CTcan complementultrasonographyinthediagnosisofacuteportalveinthrombosis,eventhoughultrasonographycanreliablyident ifyfreshthrombi[27,28].Inourstudy,theaverageageofpatients admittedwithacutePVTwas56.1years(range32-70years).Theagerangebetween51and60years old(45%)hadthehighestprevalence.Thepatientgendersplitwas65:35maletofemale.In92.5%ofthepatients,acutePVTwasf oundoncolorDopplertobepresentintheformofechogenic contentsintheportalveinandwholeorpartialabsenceofcolorflow.However,dynamicCECT revealedpartialbranchportalveinthrombosisinthe7.5%ofpatientswhohadDuplexDoppler resultsthatshowednormalcolorflowinthemainportalvein.Inallthepatientswhoshowedan absenceofflowonDuplexDoppler,acutePVTwasconfirmedonCECT.Sincepatientswith collateralswerenotincludedinthestudy,noneof theseindividualshadanyatthetimeoftheir diagnoses.

Lessthanhalf(42.5%)ofthepatientsexhibitedportalveinswithdiametersgreaterthan15mmon CECT.Three-fifthsofthepatients(35%)hadcompletethrombosis(definedbynoresidualblood flow),and65%hadpartialbloodflow.Type2a(involvementofasinglebranchoftheportalvein) wasthemostprevalent,followedbytype1(involvementoftheportalvein'strunkalone),type3(involvement ofthetrunkplusbranches),andtype2b(bothbranchesonly).Patientsmadeup37.5%,30%,17.5%,and15%ofthetotal,respectiv ely,inthisorder.Thesplenicveinwasinvolvedin thrombosisin22.5%ofthepatientsandthesuperior mesentericveinin20%.Abnormalbowelwallenhancement(hypo-enhancinggutwallormucosalhyper-enhancement)wasseenin10%ofpatients.Radiologicallydetectableasciteswaspresentin47.5%ofthepatients.Hepaticmorp hologicchangeslikeatrophy-hypertrophycomplex,nodularlivermarginsorfissuralwidening wereseeninnearlyallpatients ofCLD. Theliverwasradiologicallynormalin56%ofthenon-cirrhotics.On theother hand,TransientHepaticAttenuationDifferences(THAD)wereseenin11ofthe25non-cirrhoticpatients(44%)withdiffuseTHADshowingcentral-peripheral phenomenonbeingpredominant.Reducedenhancementinthecentreoftheliverduringthearterial phaseresultsfromdilutionwithun-opacifiedsplanchnicblood.Collateralsareabundantinthe centralorhilarregionoftheliverandintheeventofportalveinthrombosis,thisregioncontinues toreceiveportalbloodsupplyviacollaterals.Giventhatthereisarelativelysmallernumberof collaterals intheperipheryoftheliver,whenthemainportalveinflowdecreases,there isdecreased portalflowtotheperipheryoftheliver, increasedhepatic arterialflow,andaTHADasaresult.

Inthegroupthatreceivedearlytreatment,thereweremorepatientswhodemonstratedre-canalizationandfewerfatalities.Theassociationofearlytreatmentwithre-canalizationwas,however,notstatisticallysignificant(p-value=0.121).Ourfindingsareinlinewithearlierresearch byLemma A etal.[29]andAttali J etal.[30],whichindicatedthatacutePVTpatientswhoreceivedearlyanticoagulationmedicationsawre-canalizationratesof29%and30%,respectively,afterayearoffollow-up.

Conclusions:-

TheroleofvariousimagingmodalitiesismajorinacutePVT.DuplexDopplercandetectAPVTin92.5%ofpatientsandthus,isa goodscreeningtool.However,CECTshouldbeusedforconfirmationofacutePVT.Hepaticmorphologicchangesintheformo fTHADarealsoseeninnon-cirrhoticswithacutePVTwithapredominanceofdiffuseTHADwiththecentral-peripheralphenomenon.Insomewhatfewerthanhalfofthenon-cirrhoticindividualswithacutePVT,ascitesisdiscovered.Theportalveindiametermayormaynotbeincreased.Theoutcome ofacutePVTisslightlybetterinthepatientsreceivingearlyanticoagulation.

References:-

1. LauttWW,LegareDJ,D'AlmeidaMS.Adenosineasputativeregulatorofhepaticarterialflow(thebufferresponse).AmJ Physiol1985;248:H331–H338.
2. LauttWW.Thehepaticartery:subservienttohepaticmetabolismorguardianofnormalhepaticclearanceratesofhumoral substances.GenPharmacol1977;8:73–78.
3. BredfeldtJE,RileyEM,GroszmannRJ.Compensatorymechanismsinresponsetoanelevatedhepaticoxygenconsumpti oninchronicallyethanol-fedrats.AmJPhysiol1985;248:G507–G511.

4. LauttWW.Controlofhepaticarterialbloodflow:independencefromlivermetabolicactivity.AmJPhysiol1980;239:H559–H564.
5. LauttWW.Mechanismandroleofintrinsicregulationofhepaticarterialbloodflow:thehepaticbufferresponse.AmJPhysiol.1985;249:G549-56.
6. GanundaGE,BolognesiM,NeriD,etal.Preoperativeselectiveportalveinembolization(PSPVE)beforemajorhepaticresection:EffectivenessofDopplerestimationofhepaticbloodflowtopredictthehypertrophyrateofnon-embolizedliversegments.Hepatogastroenterology.2002;49:1405-11.
7. MathieuD,VasileN,DibieC,GrenierP.Portalcavernoma:DynamicCTfeaturesandtransientdifferencesinhepaticattenuation.Radiology1985;154:743–48.
8. PlattJF,RubinJM,EllisJH.Hepaticarteryresistancechangesinportalveinthrombosis.Radiology1995;196:95–98.
9. KandaH,NimuraY,YasuiA,etal.Hepaticbloodflowafteracutebiliaryobstructionanddrainageinconsciousdogs.Hepatogastroenterology1996;43:235-240.
10. TesslerFN,GehringBJ,GamesAS,etal.Diagnosisofportalveinthrombosis:valueofcolorDopplerimaging.AJRAmericanJournalofRoentgenology1991;157:293-6.
11. BatchAM,HannLE,BrownKT,etal.PortalveinevaluationwithUS:ComparisontoangiographycombinedwithCTarterialportography.Radiology1996;201:149-4.
12. ChawlaY,KumarS.PortalvenousflowinBudd-Chiarisyndrome.IndianJournalofGastroenterology:OfficialJournaloftheIndiansocietyofGastroenterology1998;17:37.
13. ShinmadaK,IsodaH,OkadaT,etal.Non-contrast-enhancedMRportographywiththtime-spatiallabelinginversionpulses:Comparisonofimagingwiththree-dimensionalhalf-fourierfastspin-echoandtruesteady-statefree-precessionsequences.JournalofMagneticResonanceImaging2009;29:1140.
14. FurutaA,IsodaH,YamashitaR,etal.Non-contrast-enhancedMRPortographywithbalancedsteady-statefree-precessionsequenceandtime-spatiallabellinginversionpulses:comparisonofimagingwithflow-inandflow-Outmethods.JournalofMagneticResonanceImaging2014;40:583-7.
15. OhnoT,IsodaH,FurutaA,etal.Non-contrast-enhancedMRPortographyandhepaticvenographywithtime-spatiallabelinginversionpulses:comparisonat1.5Teslaand3Tesla.ActaRadiologicaOpen2015;4.
16. EnglesbeMJ,KubusJ,MuhammadW,SonnendayCJ,WellingT,PunchJD,etal.Portalveinthrombosisandsurvivalinpatientswithcirrhosis.LiverTranspl2010;16:83–90.
17. BullerHR,SohneM,MiddeldorpS.Treatmentofvenousthromboembolism.JThrombHaemost2005;3:1554–60.
18. RivaN,AgenoW,SchulmanS,etal.Clinicalhistoryandantithrombotictreatmentofincidentallydetectedsplanchnicvein thrombosis:amulticentreinternationalprospectiveregistry.TheLancetHaematology2016;3:e267-75
19. HollingsheadM,BurkeCT,MauroMA,WeeksSM,DixonRG,JaquesPF.Transcatheterthrombolytictherapyforacutemesentericandportalveinthrombosis.JVascIntervRadiol2005;16:651–661.
20. ChenC.Directthrombolytictherapyinportalandmesentericveinthrombosis.JVascSurg2012;56:1124–26.
21. KlingerC,RieckenB,SchmidtA,DeGottardiA,MeierB,BoschJ,etal.Transjugularlocalthrombolysiswith/withoutTIPSinpatientswithacutenon-cirrhotic,non-malignantportalveinthrombosis.DigLiverDis2017;49:1345–1352.
22. KalliniJR,GabrA,KulikL,GangerD,LewandowskiR,ThornburgB,etal.Noncirrhoticcompleteobliterativeportalveinthrombosis:Novelmanagementusingtrans-splenictransjugularintrahepaticportosystemicshuntwithportalveinrecanalization.Hepatology2016;63:1387–90.
23. PimpalwarS,ChinnaduraiP,HernandezA,KukrejaK,SiddiquiS,JustinoH.Trans-splenicaccessforportalvenousinterventionsinchildren:Dobenefitsoutweighrisks?CardiovascInterventRadiol2018;41:87–95.
24. BlumU,HaagK,RössleM,OchsA,GabelmannA,BoosS,etal.Noncavernomatousportalveinthrombosisinhepaticcirrhosis:treatmentwithtransjugularintrahepaticportosystemicshuntandlocalthrombolysis.Radiology1995;195:153–157.
25. HanG,QiX,GuoW,NiuJ,BaiM,FanD.Transjugularintrahepaticportosystemicshuntforportalveinthrombosisincirrhosis.Gut2012;61:326–27.
26. QiX,HanG.Transjugularintrahepaticportosystemicshuntinthetreatmentofportalveinthrombosis:acriticalreviewofliterature.HepatolInt2012;6:576–90.
27. MillerVE,BerlandLL.PulsedDopplerduplexsonographyandCTofportalveinthrombosis.AmJRoentgenol1985;145:73–6.
28. CohenJ,EdelmanRR,ChopraS.Portalveinthrombosis:areview.AmJMed1992;92:173–82.
29. LemmaA,ÅbergF,MäkisaloH,VikatmaaP,MentulaP,LeppäniemiA,SallinenV.Predictorsofinsufficientrecanalizationandportalhypertensivecomplicationsaftertreatmentofnon-cirrhotic,non-malignantportalveinthrombosis-apopulation-basedstudy.ScandJGastroenterol.2020;55(11):1324-32.
30. Attali J,Heurgue A,Loock M,Thiefin G,Marcus C,Long A.Computedtomographyfollow-upofacuteportalveinthrombosis.DiagnosticandInterventionalImaging,2014; 95(6): 579-85.