Spectrum Of The Cardiac Manifestations Among Hemodialysis Patients In A Tertiary Care Hospital

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ABSTRACT

Objective: To determine Cardiac Conditions in CKD patients reported in Nephrology Department AIMS
Method: The cross sectional study was conducted at Nephrology Department Abbas Institute of Medical Sciences (AIMS) .Total 102 cases were analyzed who came from different are as suspected CVD abnormalities in Nephrology Department AIMS for Dialysis in Dialysis Unit.
Conclusion: CKD is an independent risk factor for CVD and majority of patients expire due toCVD than progress to ESRD. This risk worsens as the severity of renal dysfunctionworsens. Identification of patients with early CKD is crucial as prevention works betterthan cure. Apart from the traditional risk factors, novel risk factors peculiar to CKDresultsinearlyandrapidprogressionofCVD.Anarrayofinvasiveandnon-invasivetests is available for diagnosis of CVD but the utility of each of them in patients withCKD is still uncertain. Treatment benefits with reperfusion therapy extend to patientswith renal dysfunction but the degree of protection and risks involved needs furtherevaluation. Most of the recommendations are based on single-center data or post hocanalyses. Further randomized control trials are warranted to assess the various modalitiesforevaluation andmanagementof CVDs in CKD

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INTRODUCTION: Richard Bright firstproposed a renal etiology for cardiovascular disease (CVD)in 1836. Numerous epidemiological studies that compared patients with chronic kidney disease (CKD) to the general population and found that they had more severe and frequent CVD. Infact, those with early stages of CKD are more likely to die from CVDs than from end-stage renaldisease, and the condition itself is seen as being an analogue to coronary artery disease (CAD) (ESRD). [1] Although theseverity of this danger has been well acknowledged, people with renal impairment havebeen conveniently left out of the trials. Therefore, there is a shortage of evidence-basedmanagement of CVD in CKD. This has major treatment consequences since preventionmethods. Cardiovascular morbidity and mortality would also be avoided as CKD progressed. The idea that CKD and CVD can both cause and exacerbate one anotherresulted in the development of cardio renal

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syndrome as a distinct clinical entity. [2] Theunderlying cause ofthe elevated risk ofcardiovascular events in CKDpatients is notwell understood. Such a relationship is thought to be caused by a number of reasons.Even after accounting for the several coexisting risk variables, CKD still seems to play asignificant role in determining cardiovascular morbidity and death. [3] In this review, we outline the information that is currently known about the prevalence of CVD in CKD, risk factors, diagnostic approaches, and available therapeutic choices, along with potential future research directions. In chronic renaldisease, cardiovascular disease(CVD) is the main cause of morbidity and mortality (CKD). In stages 3 and 4 of CKD, respectively, the risk of CVD mortality doubles and triples. Each condition makes the other more common and causes it to progress, making this link complex and bidirectional. 2,3 The cardiorenal syndrome, in which the failure of one organ causes and worsens the dysfunction of theother,servesas anexample ofhow theheartandkidneyareintimatelyintertwined. [4,5] The presence of CKD and accompanying comorbidities makes it more difficult todetect andmanageCVD. CKD-CVD is additionally influenced by additionalCKD-related risk factors and alternate pathogenesis. Patients with CKD are frequently left out of clinical studies, which have a negative impact on the generalizability of evaluationmethodsandthequalityofthedatasupporting thesafetyandeffectivenessofmedications that are now available. Reviews that wereupdated in 2018 showed that CKD patients were still underrepresented, with 46–57% ofCVD studies not including them. [8,9]Recent suggestions to encourage the inclusion ofpatientswithCKDhavetakenintoaccounttheregulatoryandfinancialincentives,changestoresearch design,andcooperationbetweencardiologistsand nephrologists. If we want to get the crucial information to direct management of this vulnerablepopulation, we need to use these tactics. 10 Indeed, a significant number of problems,adverse events, and dose adjustment worries are linked to present treatment options,especially for drugs that are eliminated through the kidneys. 1 Due to clinicians' caution,CVD is frequently not adequately treated. 11 The purpose of this essay is to discuss thelimitations and present state of knowledge on CKD-CVD. It will concentrate on valvularheart disease, heart failure, arrhythmias, and coronary artery disease (CAD) (VHD) andCardiovascularDisease The presence of CKD and accompanying comorbidities makes it more difficult todetect andmanageCVD. CKD-CVD is additionally influenced by additionalCKD-relatedrisk factors and alternatepathogenesis. [1] Incidence of CAD increases linearly as GFRdrops, and it is prevalent in CKD. A difficult prognosis is brought on by the simultaneous care of CAD in these patients. [27]Lipid-lowering drugs serve as the corner stone of CAD therapy. With the advancement of CKD, statins become less helpful, and there is no obvious benefit for dialysis patients. In mild to moderate CKD, newer medicines are effective and safe, buttheir effects in advanced illness remain unknown. Although further research is required,the SHARP trial and other studies have suggested that taking ezetimibe and statins simultaneously may safely reduce cardiovascular risk, especially for people with ESKD.28,29.Changing Cardiovascular Disease Risk in ProgressingChronic KidneyDisease

LITERATUREREVIEW: Ischemicheart disease,congestiveheart failure,arrhythmias, and peripheral vascular disease are all on the CVD spectrum in CKD. We will concentrate on ischemicheart disease forthemajority of ourdiscussions becauseit istheleading cause of cardiovascular morbidity and mortality. It is clear from evidence from numerous epidemiological studies that persons with CKD are more likely to acquire CVD. [4]Patients with CKD die more frequently and earlier from CVD. At first, it was believedthat this only applied to those with end-stage renal disease (ESRD), who had a 20–30-fold increased risk of dying from CVDs compared to the general population. Similar trends were seen in the adjusted risk of hospitalization and mortality.
Similar trends in cardiovascular mortality with eGFR were found by van derVeldeetal.[6]inacollaborativemeta-analysisof10cohortsinvolving266975participants.
Albuminuria was linked to an increased risk of death

from all causes in thesame research. A linear relationship between blood creatinine levels and cardiovascularmortality was discovered in the Hypertension Detection and Follow-up Program, with a five fold difference between the lowest and highest serum creatininestratum.The 2014 United States Renal Data System.

The degree of renal impairment, the presence of proteinuria, and the rate at which these changes take place all affect the CVD risk in CKD. Those who experienced worsening renal impairment during the Multiple Risk Factor Intervention Trial were at asignificant risk for CVD. Rapid decline in renal function and co-existing proteinuria bothraise the chance of developing CVD. Irie et alfindings .'s on the separate yet combined effects of proteinuria and eGFR on the risk of cardiovascular death in a cohortofJapanese patients were further supported by Irie etal

According to the Chronic Renal Insufficiency Cohort (CRIC) study, CVD furtheraccelerates CKD's rate of development.[9] A history of heart failure was independently linked in the CRICtrial to a 29% greater chance of developing end-stage renal disease (ESRD). Additionally, it was demonstrated that people with CVD at baseline were more likely to experience deterioration in renal function in the combined analyses of the Atheroscleros is Risk in Communities Study and Cardiovascular healthstudy. Throughout the Chronic Renal Insufficiency Cohort(CRIC) research, [10].

RiskFactorsforCVDinPatients withCKD: The increased incidence of CVD in CKD is only partially accounted for by thehigherprevalenceoftraditionalriskfactorsinthesep atients.Thishasturnedourattentionon thenon-traditionalorthe 'novel' risk factorsunique to CKD.

ThetraditionalriskfactorsforCVD suchas increasingage hypertension, Dyslipidemia diabetes, smoking and obesity areriskfactorsfor CKDaswellandhencearecommoninpatients withCKD.Thenon-traditional or'novel' riskfactorsare: 'Uremia specific', or at least much more common in patients with CKD than inthegeneralpopulation. These include albuminuria Anaemia, Hyperparathyroidism, metabolicbonedisease, hyperhomocysteinaemia, malnutrition,apolipoproteinisoforms Inflammation,endothelialdysfunction Oxidativestress.The many traditional and non-traditional risk factors frequently have an additiveeffect, hastening the course of CKDand atherosclerosis. [11]

In a study of 2423 patients with CKD, Weiner et al.[13] found that the risk ofmyocardial infarction or death was four times higher in patients with anemia and LVHthan in those who had only one of these risk factors. However, the risk was only three timeshigherinpatientswithCKDwhohadneitherofthes eriskfactors.Thetwoinflammation-relatedindicatorsthataremostfrequentlymeasuredar eC-reactiveprotein (CRP) and interleukin-6. Despite the fact that CRP has been proved to be areliableindicatorof cardiovascular mortality, itsclinicalvalue is restricted.

Theroleofmicroalbuminuria: Microalbuminuria is a frequent finding in individuals with hypertension, diabetes,and dyslipidemia and is a key indicator of who is at risk of developing kidney disease[18]. Trials aiming to lessen the incidence and progression of proteinuria through theblockade of the Renin-Angiotensin-Aldosterone System (RAAS) have been successful[21]. This is because patients with diabetic nephropathy have a significantly increasedincidence of mortality with the onset of proteinuria. But after nephropathy has beenidentified, early intervention is essential for stopping the spread of the disease becauseRAAS blockage does not appear to have any positive effects. A vicious loop is alsocreated by the fact that hypertension and CKD are fundamentally related. The risk of all-cause mortality, CV mortality, acute myocardial infarction, and heart failure is higher inhypertensivesubjects withelevated pulse pressure and proteinuria[22].

DiagnosisofCVDinCKD: PatientswithCKDusuallylackt hetraditionaltriadofischemicsymptoms,increased cardiac biomarkers, and ECG abnormalities, making the diagnosis of CADdifficult. ThespecificityforthediagnosisofCADisdecreasedbyth eprevalenceofabnormalECGs;in one study, 46% of patients exhibited some abnormalities. [17] ECGs can be used tofindbundlebranchblockagesandLVH.LVmassindexa ndregionalwallmotionanomaliessuggestiveofCADare oftenmeasuredusingtwo-

dimensional(2D)transthoracicechocardiography,whichiseasilyaccessible,affordable,butoperatordependent. Due to patients' low exercise tolerance when they have CKD, exercise testingisoflittleuse.Tofindmyocardialscarring,cardiacMRIisquitehelpful.Forthedetection of coronary artery stenosismore than 70%, stress cardiacmagnetic resonanceishelpful.PatientswithCKDhavedecreasedsensitivity forsingle-photonemissionCTscanstodetectCADduetogreaterbaselineadenosinelevels. In comparison to traditional angiography,stress thallium scanning had a 62% sensitivity and 76% specificity for CA stenosis ofgreater than 75% in the Vandenberg et al. study[21].

Myocardialperfusionscintigraphy(MPS)ispreferableinpatientswithuncontrolledhypertensionandarrhythmia,whereasDSEshouldbeemployedinindividualswithreversibleairwaysdiseaseandhypotension.**Utilityofvariousdiagnosticmodalitiesfordiagnosisofcardiovasculardiseasein CKD:**The use of cTn to evaluate prognosis in patients with CKD hasproduced encouraging findings. According to a published meta-analysis, higher cTn-Twas linked to a threefold increase in all-cause mortality and cTn-I was linked to a 2.7-fold increase, rise in mortality from all causes. Regardless of the severity of renaldysfunction, an elevated cTn-T was found to be a very significant predictor of a poorshort-termprognosisintheGlobalUseofStrategiestoOpenOccludedCoronary

ManagementofCVD inCKD: The mainstay of CVD medical care is the use of aspirin, statins, ACE inhibitors(ACEi),angiotensinreceptorblockers(ARBs), andblockers.Asinthegeneralpopulation, optimal glycemic management and blood pressure control are crucial forCKD patients. Despite the fact that individuals with CKD frequently have CVDs, little isknown about the best way to treat this population. In patients with normal renal function, ACEi or ARBs constitute the standardmedical therapy. The Fosinopril in Dialysis (FOSIDIAL) trial failed to demonstrate anyadditional benefit in patients receiving dialysis, whereas the study by Efrati et al.[31]revealed a 52% decrease in mortality among dialysis patients, raising questions about thebeneficialeffectofACEi/ARBsinrenalinsufficiency. Useof ACEi/ARBs was linked to better survival in non-dialysis-dependent patients in thestudy by Molnar et al. a 50% increase in bothmajor and minor bleeding risk wasobserved. [30] Although UFH is generally safer, the risk of bleeding with UFH also risesasrenaldysfunctionbecomesmore severe.This recommendation deviates from theACC/AHArecommendationsinthedefinitionofatherosclerotiCVD(ACC/AHAusesa broader definition), the treatment of patients over 75 years of age, and the use of ahigher CVD risk level (10-year risk of 10% vs. 7.5% in ACC/AHA) when determiningstatin therapy. [36] Statin therapy should be decided on an individual basis by doctorsbecause it is unclear whether there is an eGFR limit, or the so-called "point of no return,"beyondwhich statins stop being beneficial.Anaemiamanagementisacrucialcomponentof CKDcare,andanaemiaitselfincreases the cardiovascular burden. In order to benefityourheartandbodyingeneral,therapyshouldbeadjustedtoreachthedesiredhaemoglobinrangeof 10 to 12 g/dL.

MATERIALANDMETHOD

StudywasconductedatDepartmentOf NephrologyAIMS Muzaffarabad AJ&K over 6 months.. Crosssectionalstudy. Total102Sampleswereanalyzed.samplesizecalculatedusingformulaS=Z2 ×P×(1−P)M2(1−P)M2. Datawasanalysed using SPSSsoftwareVersion 25.

DATAANALYSISANDRESULTS

FrequenciesStatistics

		Age	Gender
N	Valid	102	102
	Missing	0	0
Mean		2.9118	1.5000
Std.ErrorofMean		.08601	.05702
Median		3.0000	1.0000

Mode	3.00	1.00
Std.Deviation	.86863	.57592
Skewness	-.659	.952
Std.Error of Skewness	.239	.239

Frequency Table (AGE)

	Frequency	Percent
17to28 years	10	9.8
28to35 years	12	11.8
35to60 years	58	56.9
60to80 years	21	20.6
lessthan17 years	1	1.0
Total	102	100.0

Socioeconomic status

Sr. No	Socioeconomic Status	No of Patients
1	POOR	26
2	FAIR	30
3	GOOD	29
4	RICH	17

Durationbasecardiacconditions

Sr.No	CardiacConditions	No of patients(4 hours onceweekly)	NoofPatients(Twiceweekly 3.5hours)
1	PulmonaryHypertension	----	7(6.8%)
2	LVH	----	11(10.7%)
3	SystolicDysfunction	----	05(4.9%)
4	DiastolicDysfunction	2(1.9%)	31(30.3%)
5	Pericardialeffusion	----	21(20.5%)
6	MitralRegurgitation	1(0.9%)	41(40.1%)
7	AR(ArterialRegurgitation)	1(0.9%)	16(15.6%)
8	ArterialStenosis	----	04(3.9%)
9	TricuspidRegurgitation	2(1.9%)	40(39.2%)
10	DCMP(dilatedcardiomyopathy)	----	17(16.6%)
11	VegetationinR.A	----	03(2.9%)
12	Ischemic Dilatedcardiomyopathy	----	05(4.9%)
13	Vegetationinaorticvalve	----	01(0.9%)
14	SevereConcentricLVH	----	02(1.9%)
15	SWMA(segmental wall motionabnormality)	----	03(2.9%)
16	SitusSolitis	----	04(3.9%)
17	Levocardia	----	09(8.8%)
18	Biarterialenlargement	----	03(2.9%)
19	RestrictiveCardiomyopathy	----	04(3.9%)
20	Pulmonaryregurgitation(PR)	----	07(6.8%)
	TotalPatients	6	102

INTRA DIALYTIC COMPLICATIONS BASEDCARDIACCONDITIONS

CardiacConditions	No of	No of	No	No	No	No

	patients(Hypotension)	patients(Hypertension)	ofpatients(Cramps)	ofpatients(Nausea)	ofpatients(Headache)	ofpatients(Chest-pain)
PulmonaryHypertension	01(0.9%)	1(0.9%)	3(2.9%)	3(2.9%)	1(0.9%)	2(1.9%)
LVH	04(3.9%)	2(1.9%)	3(2.9%)	1(0.9%)	3(2.9%)	2(1.9%)
SystolicDysfunction	01(0.9%)	----	1(0.9%)	1(0.9%)	3(2.9%)	1(0.9%)
DiastolicDysfunction	12(11.9%)	6(5.9%)	9(8.9%)	7(6.8%)	12(11.7%)	4(3.9%)
Pericardialeffusion	13(12.7%)	1(0.9%)	4(3.9%)	2(1.9%)	08(7.8%)	2(1.9%)
MitralRegurgitation	21(20.5%)	4(3.9%)	9(8.9%)	3(2.9%)	19(18.6%)	1(0.9%)
AR(ArterialRegurgitation)	06(5.9%)	1(0.9%)	3(2.9%)	1(0.9%)	9(8.8%)	5(4.9%)
ArterialStenosis	01(0.9%)	----	----		1(0.9%)	2(1.9%)
TricuspidRegurgitation	19(18.6%)	4(3.9%)	4(3.9%)	3(2.9%)	13(12.7%)	1(0.9%)
DCMP(dilatedcardiomyopathy)	01(0.9%)	----		2(1.9%)	1(0.9%)	----
VegetationinR.A		----	1(0.9%)	1(0.9%)	1(0.9%)	----
Ischemic Dilatedcardiomyopathy	----	----	1(0.9%)	1(0.9%)	1(0.9%)	----
Vegetationinaorticvalve	----	----	3(2.9%)	3(2.9%)	1(0.9%)	----
SevereConcentricLVH	----	----	1(0.9%)	01(0.9%)	1(0.9%)	----
SWMA(segmentalwall motionabnormality)	02(1.9%)	----	4(3.9%)	5(4.9%)	----	----
SitusSolitis	-----	----	----	----	----	----
Levocardia	----	----		----	1(0.9%)	
Biarterialenlargement	1(0.9%)	----	----	----	1(0.9%)	1(0.9%)
RestrictiveCardiomyopathy	1(0.9%)	----	----	----	1(0.9%)	2(1.9%)
Pulmonaryregurgitation(PR)	----	----	----	----	----	1(0.9%)

HemoglobinLevelsBasedCardiacConditions

CardiacConditions	Hb(<8g/dL) n= 12	Hb(8 to 10 g/dL) n= 36	Hb(10to 11.5 g/dL)n=28	Hb(>11.5 g/dL) n=26
PulmonaryHypertension	1(0.9%)	3(2.9%)		1(0.9%)
LVH	4(3.9%)	4(3.9%)	2(1.9%)	
SystolicDysfunction		5(4.9%)		
DiastolicDysfunction	5(4.9%)	19(18.6%)	5(4.9%)	4(3.9%)
Pericardialeffusion	6(5.8%)	18(17.6%)		2(1.9%)
MitralRegurgitation	6(5.8%)	30(29.4%)	5(4.9%)	3(2.9%)
AR(ArterialRegurgitation)	4(3.9%)	11(11.7%)	3(2.9%)	
ArterialStenosis		3(2.9%)		
TricuspidRegurgitation	7(6.8%)	26(25.4%)	4(3.9%)	5(4.9%)

DCMP(dilatedcardiomyopathy)		2(1.9%)		
VegetationinR.A	2(1.9%)	1(0.9%)	1(0.9%)	
Ischemic Dilatedcardiomyopathy	1(0.9%)	1(0.9%)		
Vegetation inaorticvalve				1(0.9%)
SevereConcentricLVH	2(1.9%)	2(1.9%)	2(1.9%)	
SWMA(segmentalwall motionabnormality)		3(2.9%)	3(2.9%)	
SitusSolitis	1(0.9%)	1(0.9%)	1(0.9%)	
Levocardia	1(0.9%)	1(0.9%)		
Biarterialenlargement	1(0.9%)	1(0.9%)		
RestrictiveCardiomyopathy	1(0.9%)	1(0.9%)		
Pulmonaryregurgitation(PR)	2(1.9%)	2(1.9%)		

DISCUSSION

Inchronicrenaldisease,cardiovasculardisease(CVD)isthemaincauseofmorbidity and mortality (CKD). In stages 3 and 4 of CKD, respectively, the risk of CVD mortality doubles and triples. Each condition makes the other more common and causes it to progress, making this link complex and bidirectional. 2,3 The cardiorenal syndrome,in which the failure of one organ causes and worsens the dysfunction of the other, servesasan example of how theheartand kidneyareintimatelyintertwined. [4,5]

The presence of CKD and accompanying comorbidities makes it more difficult todetect andmanageCVD. CKD-CVD is additionally influenced by additionalCKD-related risk factors and alternate pathogenesis. Patients with CKD are frequently left outof clinical studies, which has a negative impact on the generalizability of evaluationmethodsandthequalityofthedatasupporting thesafetyandeffectivenessofmedications that are now available. According to systematic studies published in 2006,56-75% of CVD trials did not include participants with CKD. Patients with advancedkidney disease had an even higher percentage of this (ESKD).[6,7] Reviews that wereupdated in 2018 showed that CKD patients were still underrepresented, with 46–57% ofCVD studies not including them. [8,9]Recent suggestions to encourage the inclusion ofpatientswithCKDhavetakenintoaccounttheregulatoryandfinancialincentives,changestoresearch design,andcooperationbetweencardiologistsand nephrologists.

If we want to get the crucial information to direct management of this vulnerablepopulation, we need to use these tactics. 10 Indeed, a significant number of problems,adverse events, and dose adjustment worries are linked to present treatment options,especially for drugs that are eliminated through the kidneys. 1 Due to clinicians' caution,CVD is frequently not adequately treated. 11 The purpose of this essay is to discuss thelimitations and present state of knowledge on CKD-CVD. It will concentrate on valvularheart disease, heart failure, arrhythmias, and coronary artery disease (CAD) (VHD) andCardiovascularDiseaseThepresence of CKDand accompanyingcomorbiditiesmakesitmore difficult to detect andmanageCVD. CKD-CVD is additionally influenced by additionalCKD-relatedrisk factors and alternatepathogenesis. [1]

PatientswithCKDarelargelyomittedfromclinicalresearch,whichhasanegative impact on the generalizability of evaluation methods and the quality of the datasupporting the safety and effectiveness of medications that are now available. Accordingtosystematicstudiespublishedin2006,56-75%ofCVDtrialsdidnotincludeparticipantswithCKD.Patientswithadvancedkidneydiseasehadanevenhigherpercentage of this (ESKD). [6,7] Reviews that were updated in 2018 showed that CKDpatients were still underrepresented, with 46–57% of CVD studies not including them.[8,9]Wehavestudied102patientsofundergoing hemodialysisindialysiscenterAIMSMZD.

Inthistenurepatientsfromchildrentooldagewerekeen

lyobserved&interviewed.Amongthemweremale(about54%), and 47were females (46%). Inourstudytheprobabilityofcardiacissuesin CKD-5dpatientsisseentobe 100%. Wehaveconcludedmanyreasonsbehind cardiacissues/conditionssuchas, Hypertension, Lackofawareness, CardiacstressduringHD procedure PoorHealthconditionssuchasanemia Othercomorbs Issues related to vascular accessElectrolytesimbalancedueto : InadequateHDbecauseofpatientandhospitalfactors Poorcompliancetomedicationsbecauseoffinancialissues Hypoparathyroidism Chronicfluidoverloadconditions HyperphosphatemiaandincreasedCa×po4product

CONCLUSION: CKD is an independent risk factor for Cardio Vascular Disease (CVD) and is acauseofincreasedmortalityinCKD.Thisriskworsensastheseverityofrenaldysfunctionworsens.Identificationofpatientswithearly CKDiscrucialasprevention works better than cure. Apart from the traditional risk factors, novel risk factors peculiarto CKD results in early and rapid progression of CVD. An array of invasive and non-invasive tests is available for diagnosis of CVD but the utility of each of them in patientswithCKDisstilluncertain.Treatmentbenefits withreperfusiontherapyextendtopatients with renal dysfunction but the degree of protection and risks involved needsfurther evaluation. Most of the recommendations are based on single-center data or posthocanalyses.Furtherrandomizedcontroltrialsare warrantedtoassessthevariousmodalitiesfor evaluation andmanagementofCVDs in CKD.

RECOMMENDATIONS: Engage the patient in healthyactivities**,** Counsel the patient & spread awareness**,** Health department should arrange awareness sessions for public**,** Seminars and workshops should be arranged for public. Training of dialysis technicians and doctors anddoctors for preventing & identifying the cardiac stresses related to CKD**,** Free provision of iron supplements and erythropoietin to combat the issues ofanemialeading to CVD.Provision of free medicines to control Blood pressure and maintain optimumlevelsof calcium, phosphorous and PTH levels. Regular visits of doctors by increasinghuman resources and training of dialys is technician for assessment of target weight of patients. Installation of dialys is units in remote area swith sufficient facilities and human resource to decrease patient related factors leadingto inadequate hemodialysis

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