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# ECHOCARDIOGRAPHIC FINDINGS IN CHRONIC KIDNEY DISEASE (CKD) PATIENTS

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**ABSTARCT**
Cardiovascular diseases are the cause for mortality in population. The early diagnosis is important in the management of these diseases and to prolong the lifespan. Anemia, Arterio-venous communications, fluid and electrolyte imbalances and nutritional disturbances in patients with End Stage Renal Disease (ESRD) may also contribute to cardiac decompensation .In the general population Cardio vascular Diseases (CVD) morbidity and mortality have declined substantially over the past three decades through risk factor identification and reduction and more effective treatment of coronary artery disease. To estimate the levels of serum creatinine and blood urea to identify the ESRD and to assess the levels of LV function and detect pericardial abnormalities in patients with advanced renal insufficiency by Echo and the levels of serum creatinine and Echo findings are compared with normals and ESRD patients. **Group – I:** Healthy normal individuals not having diabetes mellitus, hypertension, kidney diseases aged between 20-60yrs and not having habits like alcohol ingestion and smoking. **Group-II:** Subjects patients attending Nephrology out patient department (O.P.D) for dialysis are selected. Diagnosis of CKD is confirmed by Nephrologists. Patients with arterio venous fistula, Diabetes mellitus, Known case of Coronary Artery Disease (CAD), Valvular heart disease, Congenital heart disease, Patients on treatment with erythropoietin, Poor echo window, Patients with history of smoking and alcohol ingestion. **Results** .Serum creatinine levels indicate eGFR and the values indicate decrease in glomerular function Echo findingns in almost all the patients indicate significant changes in LV thickness,volume,velocity of shortening. It is observed in the present study blood pressure is increased due to fluid over load and this may be due to release of plasma catecholamines which may increase one's risk of developing hypertension.

**KEYWORDS**: CKD, chronic, heart failure, ECHO.

## INTRODUCTION

Cardiovascular diseases are the cause for mortality in population. The early diagnosis is important in the management of these diseases and to prolong the lifespan. The number of investigative procedures both invasive and non-invasive are used by cardiologists.

Most of the CVD are as a result of complication of metabolic disorders like DM, hyperlipidemia, atherosclerosis, hypertension, valvular diseases, rheumatic fever, endocrinal causes and renal causes., Renal diseases are most common as a complication of DM or nephritis or renal vascular diseases .The diagnosis of these are often at early stages of renal failure .The stages of renal failure are diagnosed by serum creatinine levels.

**End-Stage Renal Disease (ESRD):** ESRD is a progressive loss of function of more and more nephrons that gradually decreases over all kidney function.[5] The eGFR value is <15ml/min in this condition. It is also called as CRF or CKD.

The incidence of CRF varies widely by state and country. In united states the incidence is 331 new cases per million people.[35] Everyday 33 patients are undergoing dialysis in the Nephrology unit and the frequency is more. The management of ESRD is by dialysis technique.

Myocardial and pericardial diseases often cause morbidity and death in patients with ESRD. Specific uremic cardiomyopathy may develop in patients with

advanced renal insufficiency. There is also evidence that uremic serum may have a depressant action on the myocardium.While the existence of uremic cardiomyopathy as a discrete entity is moot patients with advanced renal insufficiency do have an increased incidence of CAD and hypertension both before and after they begin hemodialysis therapy. These conditions may lead to abnormal LV function and eventually to overt congestive failure.

Anemia, arterio-venous communications, fluid and electrolyte imbalances and nutritional disturbances in patients with ESRD may also contribute to cardiac decompensation.[55]

In the general population CVD morbidity and mortality have declined substantially over the past three decades through risk factor identification and reduction and more effective treatment of coronary artery disease.

Regular screening of cardiac complications by Echocardiography is the clinical procedure followed by Nephrologists in CKD patients with dialysis.

## MATERIALS AND METHODS

### Selection of Subjects

**Group – I:** Healthy normal individuals not having diabetes mellitus, hypertension, kidney diseases aged between 20-60yrs and not having habits like alcohol ingestion and smoking.

**Group-II:** Subjects patients attending Nephrology out patient department (O.P.D) for dialysis are selected. Diagnosis of CKD is confirmed by Nephrologist.

**EXCLUSION CRITERIA:** Patients with arterio venous fistula, Diabetes mellitus, Known case of CAD, Valvular heart disease, Congenital heart disease, Patients on treatment with erythropoietin, Poor echo window, Patients with history of smoking and alcohol ingestion.

### SPECIMEN COLLECTION AND HANDLING SERUM

**Serum:** Draw blood in to a clean tube allow the blood to clot for at least thirty minutes at room temperature. Separate the serum by centrifugation and store in a refrigerator or store frozen for extended use.

### ESTIMATION OF CREATININE

**Method :** Serum creatinine was measured by Jaffe's method.[8]

**Principle:** Creatinine + Picrate $\xrightarrow{OH^-}$ Janovki complex (red colour)

### ESTIMATION OF UREA:

**Method:** Blood urea was measured by Berthelot method.[23]

**Principle:** $2[NH_2 - Co - NH_2]$ urea + $H_2o \xrightarrow{urease} 2\ NH_4 + Co_2$

$NH_3$ + salicylate +Hypochlorite $\xrightarrow{Nitroprusside}$ 2,2, dicarboxyindophenol.

### ESTIMATION OF HEMOGLOBIN

**Method:** Hb is estimated using sahli's haemoglobinometer[32]

**eGFR:** eGFR can be calculated by the formula. GFR can be estimated from serum creatinine levels by using prediction equations. Cock croft – Gault equation and MDRD equations provide useful estimate of GFR in adults. But in this study cock croft Gault formula is used.[15]

$$Ccr\ (ml/min) = \frac{140 - (Age \times Weight)}{72 \times Scr} \quad \text{(if male)}$$

$$Ccr\ (ml/min) = \frac{140 - (Age \times Weight)}{72 \times Scr} \times 0.85 \quad \text{(if female)}$$

Echocardiography is done by the cardiologist of the institute. From their report the following parameters are selected for study.

**Height:** Height was recorded using the scale and expressed in centimeters.

**Weight:** Weight of all the subjects was measured by using weighing machine and expressed in kilograms.

**Body Mass Index:** The body mass index was derived by Quetelet's index.

$$\frac{\text{Body weight (kg)}}{\text{Height } (m^2)}$$

**Body surface Area (BSA):** It can be obtained from namogram chart by using height and weight. It is expressed in Sqm.

**Fractional shortening (FS):** It can be obtained from LVIDd & LVIDs by using the formula. It is expressed in %.

$$FS = \frac{LVIDd - LVIDs}{LVIDd} \times 100$$

**Ejection fraction (EF):** It can be obtained from LVEDV and LVESV by using the formula. It is also expressed in %

$$EF = \frac{LVEDV - LVESV}{LVEDV} \times 100$$

**Stroke volume (SV):** It can be calculated by using LVEDV and LVESV by the formula. It is expressed in the units ml.

$$SV = LVEDV - LVESV$$

**Cardiac output (CO):** It can be calculated by using stroke volume and heart rate. It is expressed in lit/min

CO=SV X HR

Body surface area

**Cardiac Index (CI):** It can be calculated by using cardiac output and body surface area. It is expressed in lit/min/$m^2$

$$CI = \frac{\text{Cardiac output}}{\text{Body surface area}} \times 100$$

**RESULTS**

**Table – I: Comparision of Age, BSA, BMI of controls and subjects**

| | Controls | | Subjects | | 't' | 'p' |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| **AGE (yrs)** | 43.85 | 10.73 | 46.05 | 10.23 | 0.66 | 0.51 |
| **BSA (sqm)** | 1.67 | 0.20 | 1.60 | 0.15 | 1.25 | 0.22 |
| **BMI ($kg/m^2$)** | 25.03 | 3.85 | 22.34 | 4.47 | 2.03 | 0.048 |

Statistical analysis indicates no significance in age, BSA of controls and subjects. There is statistical significance in BMI of controls and subjects with mean values lower than controls.

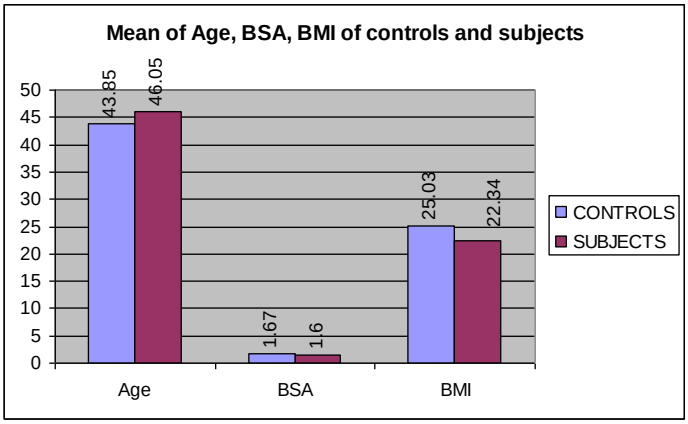



**Table – II: Comparision of HR,BP values in controls and subjects.**

| | Controls | | Subjects | | 't' | 'p' |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| **HR (beats/min)** | 72.55 | 2.68 | 90.25 | 7.39 | 10.06 | <0.001 |
| **SBP (mm of Hg)** | 115.15 | 5.52 | 155.25 | 13.71 | 12.13 | <0.001 |
| **DBP (mm of Hg)** | 77.00 | 4.70 | 86.00 | 10.95 | 3.37 | 0.0016 |

The mean values of HR, SBP and DBP values are higher than controls. 'p' value indicates significance in changes between controls and subjects.

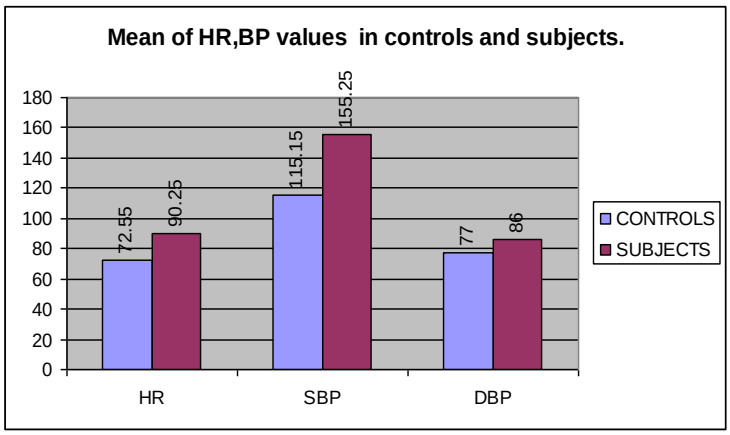


**Table – I11: Comparison of blood urea, serum creatinine eGFR and Hb among controls and subjects.**

| | Controls | | Subjects | | 't' | 'p' |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| **Blood urea** | 29.20 | 4.47 | 101.50 | 24.66 | 12.90 | <0.001 |
| **Serum creatinine** | 1.10 | 0.17 | 7.42 | 2.20 | 12.80 | <0.001 |
| **eGFR** | 75.77 | 29.34 | 10.64 | 5.72 | 9.74 | <0.001 |
| Hb | 14.18 | 1.48 | 9.14 | 1.60 | 10.34 | <0.001 |

The mean values of blood urea, serum creatinine higher in subjects than controls and statistically significant. Mean value of eGFR is much less in subjects and is statistically significant. This indicates renal failure. The higher values of blood urea, serum creatinine corresponds to low value of eGFR in subjects. The low values of Hb in subjects compared to normal values in controls are statistically significant.

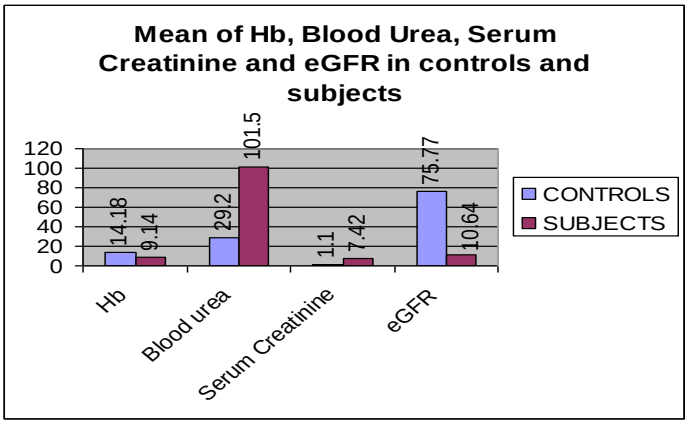



**Table – 1V:Comparision of Echo cardiography findings of left ventricle among controls and subjects.**

| | Controls | | Subjects | | 't' | 'p' |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| **LVIDd (mm)** | 43.10 | 2.43 | 47.50 | 4.64 | 3.07 | 0.0039 |
| **LVIDs (mm)** | 27.90 | 2.85 | 32.05 | 4.21 | 3.65 | <0.001 |
| **IVSDd (mm)** | 9.60 | 0.88 | 14.45 | 1.75 | 11.27 | <0.001 |
| **IVSDs (mm)** | 15.00 | 1.21 | 19.10 | 2.20 | 7.30 | <0.001 |
| **PWDd (mm)** | 9.90 | 0.97 | 14.25 | 1.62 | 10.30 | <0.001 |
| **PWDs (mm)** | 14.90 | 1.33 | 19.10 | 1.94 | 7.98 | <0.001 |
| **EDV (ml)** | 88.40 | 14.49 | 123.25 | 32.99 | 4.32 | <0.001 |
| **ESV (ml)** | 30.65 | 8.16 | 52.41 | 18.87 | 4.73 | <0.001 |

The mean values of all parameters in controls are with in normal limits and in subjects are higher compared to controls. All these changes are statistically significant. So, these changes indicate hypertrophy of left ventricle with increase in thickness of walls and volume.

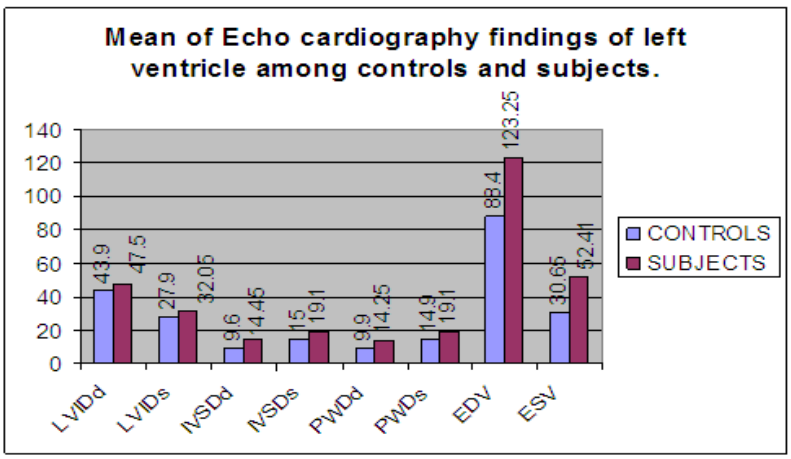
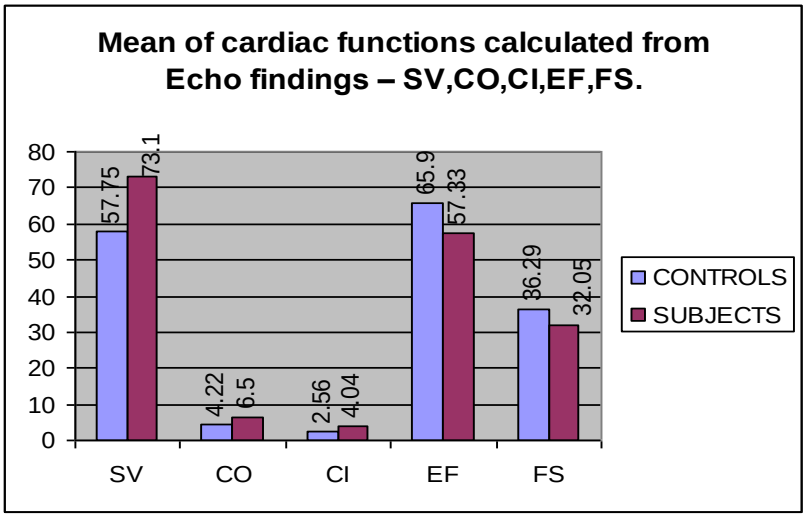


**Table –V: Changes in cardiac functions calculated from Echo findings – SV,CO,CI,EF,FS.**

| | Controls | | Subjects | | 't' | 'p' |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| **SV (ml)** | 57.75 | 10.55 | 73.10 | 21.17 | 2.90 | 0.006 |
| **CO (lit/min)** | 4.22 | 0.87 | 6.50 | 1.97 | 4.73 | <0.001 |
| **CI (lit/min/m$^2$)** | 2.56 | 0.60 | 4.04 | 1.19 | 4.96 | <0.001 |
| **EF (%)** | 65.90 | 6.31 | 57.33 | 10.55 | 3.11 | 0.0034 |
| **FS (%)** | 36.29 | 5.60 | 32.05 | 5.35 | 2.44 | 0.019 |

The mean values of SV,CO,CI are higher in subjects compared to the controls and the 'p' value indicates significant. EF, FS are less in subjects compared to controls and this change are significant.




**DISCUSSION**

In the present study 18 patients in stage -5 CRF, and 1 patient in stage-4 CRF, 1 patient in stage-3 CRF. Staging of CRF is described by the eGFR valves , serum creatinine levels, blood urea levels indicate all the patients are in ESRD. All are undergoing dialysis alternate days for the past 6 years. The reduce in GFR causes decrease in renal excretion of water and solutes and many of the waste products of metabolism such as urea and creatinine accumulate largely due to reduction in functional nephrons. As indicated in the results increase in the levels of urea and creatinine are also supported by the studies of Dangri.P et al (2003).[16] As indicated in the results Hb levels are much less in CRF patients. All the patients are suffering from anemia. Same results are observed in the studies of Robert N.Foley et al (2000) .[56].There will be decreased renal secretion of erythropoietin which leads to diminished red blood cell production and consequent anemia. In anemia, two peripheral effects greatly increased the total peripheral resistance. Those are reduced viscosity of the blood resulting from the decreased concentration of red blood cells. Other is diminished delivery of oxygen to the tissues because of the decreased Hb which causes vasodilatation. As a consequent the CO increases greatly. There is significant increase in HR of CRF patients in this study. This change may be due to the cardiovascular reflexes. In renal failures fluid retention increases venous return with raise in right arterial pressure. This stimulates the right atria stretch receptors. This initiates Bainbridge reflex response. This stimulus may be mechanical resulting from stretching of the SA node it increases the HR. Increase of both SBP and DBP valves in CRF patients are significant in the present study. The same results are also indicated in the studies of Dangri.P et al (2003).[16] The determination of SBP is stroke volume. The pre-load (LVEDV) and after load (total peripheral resistance) maintain the stroke volume. In the Echo findings indicate the increase in LVEDV. So, preload may be the cause for raise in SBP. Fluid retention increases peripheral resistance and may be the cause for raise in DBP in the present study. Increased CO raises the arterial pressure. The increased CO increases the capillary pressure so that fluid begins to transude out of capillaries in to the tissues. Increased pressure in the veins reduces the mean systemic pressure. The excess blood flow through the peripheral tissues cause autoregulatory increase in the peripheral resistance, thus increasing the resistance to venous return. These factors cause the mean systemic filling pressure to return back toward normal and the resistance vessels of the systemic circulation to constrict. Four main structural abnormalities of the heart have been described in patients with CRF. LV hypertrophy, expansion of the non vascular cardiac interstitium leading to inter myocardiocytic fibrosis, changes in vascular architecture and, myocardial calcification All these abnormalities promote systolic as well as diastolic LV dysfunction which predispose to symptomatic heart failure which is a risk factor for death. Left ventricular hypertrophy and

CAD are the strongest independent predictors of adverse cardiovascular events. LVH is a major echocardiographic findings in uremic patients. Myocardial and pericardial diseases often cause death inpatients with ESRD. There is also evidence that uremic serum may have a depressant action on the myocardium. Patients with advanced renal insufficiency do have an increased incidence of CAD and hypertension both before and after they begin hemodialysis. These conditions can lead to abnormal left ventricular function and eventually to overt congestive failure. It seems possible that heart failure or pulmonary congestion in ESRD patients who have well preserved ventricular function may be a result of the high filling pressure required to fill relatively thick walled and stiff ventricles. The inability of patients with ESRD to excrete salt and water may contribute to pulmonary congestion. There will be profound changes in $K^+$ and acid base homeostasis are frequent in patients with ESRD. Acidosis resulting from failure of the kidneys to rid the body of normal acidic products. Production of vit – D and retention of phosphate causes osteomalacia. Increased renal vascular resistance Anemia and hypertension however appear to be the risk factors most consistenly associated with this progression both in observational studies and in controlled clinical trials. Both risk factors are associated with cardial outcomes in ESRD. These factors were associated with cardiac failure a pre lethal occurrence that predated two thirds of all dialysis patient deaths. These observations suggest that risk factors like anemia and hypertension may exert their adverse impact through the follwoign maladaptive cascade risk factor, progression of LV echocardiographic abnormalities, cardiac failure, death. Hypertension is caused by excessive aldosterone secretion which increases sodium reabsorption mainly in the cortical collecting tubules.Long term mechanisms eventually develops a feed back gain for control of arterial pressure.

**Echo findings**

Echocardiography provides an excellent non-invasive method to study the details of anatomy of cardiac chambers, wall dimensions, valve movements and is also used to assess the cardiac performance. So, in the present study diameters of LV in diastole and systole are increased in CRF patients The same results are also observed in the studies of Dangri.P et al (2003).[16] And this indicates the thickness of ventricular walls. So, it is observed that the EDV is much increased in CRF patients. These results indicate LV hypertrophy. It may be due to the increase in workload on heart. For work load two factors contribute. Venous return:- Increase in mean circulatory filling pressure raises the pressure gradient for venous return. Increased pressure gradient for venous return elevates cardiac output. Increase in peripheral resistance. The present study indicates raise in ESV in renal failure. This may suggest the early stage of cardiac failure. EF and FS of LV are decreased in CRF patients. The results are similar to the studies of Robert.N.Foley et al (2000).[56] Fractional shortening fell by 8% in those destined to develop cardiac failure, compared with 0% in those without cardiac failure. The data of Robert E. Kleiger et al (1981)[55] suggest that hypertrophy and EF appears to be the first detectable abnormality. Majority of the patients also have decreased LV compliance. From observations in this study role of dialysis treatment is not important to limit the cardiovascular complications. Early diagnosis of cardiovascular changes is necessary to control the complications in chronic kidney disease patients with hemodialysis.

**CONCLUSION**

Most of the patients in the study are in End-stage renal failure. They are on hemodialysis for few months.Serum creatinine levels indicate eGFR and the values indicate decrease in glomerular function.

In dialysis the dialyzed fluid composition is same as plasma except ,urea ,glucose, $k^+$.so, cardiac complications ae common with uremia and changes in serum k+level. Accumulation of urea in high content lead to pericarditis. Hyperkalemia leads to cardiac arrythmias. There will be an increased cardiac contractility due to increased ionized calcium. Decrease in erythropoietin potentially leading to anemia which causes fatigue. vascular calcification that further impairs cardiac function.

Echo findingns in almost all the patients indicate significant changes in LV thickness, volume, velocity of shortening.

It is observed in the present study blood pressure is increased due to fluid over load and this may be due to release of plasma catecholamines which may increase one's risk of developing hypertension.

Statistical analysis indicate these changes are significant compared to normal.

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