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Estimation of levels of Creatine Kinase-MB (CK-MB) and Troponin I in Chronic Kidney Disease (CKD) patients on haemodialysis with NO history of coronary artery disease.

The details of the authors as given below:

Dr. N. Augustiya* MD Biochemistry, Assistant Professor,Biochemistry , Agaspriya127@gmail.com, 6382108968, Department of Biochemistry, Sri Manakula Vinayagar Medical College and Hospital, Madagadipet, Puducherry. PIN-605107,

Dr. Nilaya Patil, MD Biochemistry, Assistant Professor, (Biochemistry), Nilaya2005@gmail.com, 8308822597, Department of Biochemistry, Seth Gordhandas Sunderdas Medical College and the King Edward Memorial Hospital, Parel, Mumbai. Maharashtra- 400012

Dr.Leela Gul Abichandani, Head of Department, (Biochemistry), Head of Department, (Biochemistry) , Leela.abichandani@yahoo.com, 9820900807, Department of Biochemistry, Mahatma Gandhi Mission Institute of Health Sciences, MGM Campus, Kamothe, Panvel, Navi Mumbai, Maharashtra 410209

*- Corresponding author

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Abstract

Background: Chronic Kidney Disease (CKD) is a global public health issue and is becoming more common. It is an important contributor to morbidity and mortality. In India 0.68 % of people are suffering from any sort of renal disease (not CKD) and 0.16 % of people are victims of CKD. Patients with CKD have higher risk of cardiac events, which increases as the disease progress to end stage renal disease(ESRD).

Objectives: To estimate Creatine Kinase-MB (CK-MB) and Troponin I in patients undergoing haemodialysis without any history of coronary complication for earlier detection of Cardiovascular disease (CVD).

Methodology: It was a single centre hospital based cross sectional study carried out in collaboration with the Departments of Biochemistry, Nephrology and Medicine. The estimated sample size was 90 patients (30 patients in 3 groups). The groups were Group AControl group, Group B-CKD patients not on hemodialysis and Group C-CKD patients on hemodialysis

Results: A majority, 61.1% patients belonged to the age group 45-60 years. On comparison of means of CK-MB & Troponin I among the groups, patients on hemodialysis had a higher mean than patients who were not on hemodialysis.

Conclusion: There is a definitive increase in CK-MB & Troponin I values in patients with CKD on hemodialysis without any history of coronary complication. We can infer that CK-MB & Troponin I testing can be used as marker for all patients undergoing hemodialysis without coronary complication for earlier detection of CVD.

Keywords: Chronic Kidney Disease, CK-MB, Hemodialysis, Troponin I.

Introduction:

The kidney is one of the most highly differentiated organs in the body. At the conclusion of embryologic development, nearly 30 different cell types form a multitude of filtering capillaries and segmented nephrons enveloped by a dynamic interstitium. This cellular diversity modulates a variety of complex physiologic processes. Endocrine functions, the regulation of blood pressure and intraglomerular hemodynamics, solute and water transport, acid-base balance, and removal of drug metabolites are all accomplished by intricate mechanisms of renal response. (1)

Filtration and excretion of metabolic waste products (urea and ammonium), management of required electrolytes, fluid and acid-base balance, red blood cell synthesis are all critical activities of the kidneys. They also govern water reabsorption and maintain intravascular volume by regulating blood pressure through the renin-angiotensin- aldosterone pathway. The kidneys also reabsorb glucose and amino acids, and they regulate hormones through the activation of erythropoietin, calcitriol, and vitamin D. Renal blood flow normally drains ~20%

of the cardiac output, or 1000 mL/min. Blood reaches each nephron through the afferent arteriole leading into a glomerular capillary where large amounts of fluid and solutes are filtered to form the tubular fluid. (2)

Chronic kidney disease (CKD), also known as chronic renal failure (CRF), is a term that refers to all levels of impaired kidney function, ranging from mild, moderate, and severe chronic kidney failure. CKD is a global public health issue. (3)

In 2017, there were 697.5 million cases of CKD (all stages) reported worldwide, resulting in a global prevalence of 9.1%. The global all-age prevalence of CKD grew by 29.3% between 1990 and 2017.(5) In India: currently 115.1 million cases. The actual disease burden of CKD/end stage renal disease (ESRD) in the Indian population cannot be reliably determined due to absence of a renal registry. According to a trust in South India's domiciliary CKD screening programme, the prevalence of CKD stage 5 is 0.87 per thousand people.

Diabetic nephropathy was responsible for nearly one third of CKD-related disability- adjusted life years (DALYs). (6)

A higher risk of cardiovascular disease is linked to end-stage renal disease (ESRD). Chronic kidney disease has a number of unfavorable consequences for the cardiovascular system. Hemodialysis results in significant intravascular volume and electrolyte changes. HD-related cardiovascular ailments such as intradialytic hypotension (IDH) and myocardial shocking result, putting patients at risk for acute ischemic syndromes, arrhythmias, and sudden cardiac death (SCD). Cardiovascular mortality increases as CKD advances, eventually becoming the leading cause of death in ESRD patients. (7)

Patients with CKD have a higher risk of cardiac events, which increases as the disease progresses to ESRD. However, both patients and clinicians may be unaware of the elevated risk associated with both the commencement and each of HD session. As a result, a greater understanding of the issues is required, as is the development of population and patientspecific approaches to early prevention and identification of at- risk individuals. (8)

The enzymes currently in clinical use to detect myocardial infarction are CK and its isoenzyme CK-MB. CK is a cytosolic enzyme transfers high-energy phosphate between creatine and

adenosine diphosphate. CK is a dimer, each with a molecular weight of about 40 kDa; composed of two sub- units, B and M. CK-MB (CK-2) - most specific for the myocardium.(4).

CK-MB is a valuable tool for the diagnosis of MI because of its relative high specificity for myocardial damage. CK-MB activity assays have been replaced by CK-MB mass assays to measure CK-MB. CK-MB mass assays can detect an increased amount of serum CK-MB from about 1 hour. The preferred biomarkers for assessment of myocardial necrosis are the cardiac troponins. Troponin is a complex of three proteins that bind to the thin filament (actin) of cardiac and skeletal muscle. Following injury to skeletal or heart muscle cells, the troponin complex and free troponin subunits are released into the bloodstream.(4). Even in the absence of clinically diagnosed acute myocardial ischemia, patients with renal insufficiency may have elevated serum troponins. In patients with renal failure, serum troponin T is elevated more frequently than troponin I, prompting doctors to challenge its specificity for the diagnosis of myocardial infarction.(9) Hence more evidence is required to establish that CKD patients on hemodialysis are at a higher risk of Cardiovascular events.

Aim:

To estimate the levels of CK-MB and Troponin I in CKD patients on haemodialysis without any history of previous coronary artery disease.

Objective:

- To estimate the level of CK-MB and Troponin I in CKD patients who are on hemodialysis and compare it with CKD patients who are not hemodialysis and also with a control group which consisted of normal healthy individuals.

Methodology:

Study design:

It was a single center, hospital based cross sectional study.

Study setting:

The study was carried out in the Department of Biochemistry of a tertiary care hospital in collaboration with Departments of Nephrology and Medicine. The blood samples were collected from the patients admitted in the Nephrology unit and were tested for cardiac markers in the Biochemistry laboratory.

Study duration:

The study was conducted for a period of 18 months.

Study population:

Adult CKD patients aged 18 to 60 years who were in the earlier stages of kidney diseases i.e., stages 1, 2 and 3, those CKD patients who were in the final stages of chronic kidney disease i.e., stages 4 and 5 who were visiting the Nephrology unit for hemodialysis and those patients aged 18 to 60 years who were attending the Medicine Out Patient Department for illnesses other than cardiovascular diseases and kidney diseases were included in the study.

Sample size and sampling strategy:

A total of 90 patients were included in the study. The sample size was calculated using OpenEpi version 3. From previous literature it was found that around 28% of the patients undergoing hemodialysis had an elevated levels of CK-MB and troponins. Assuming the confidence interval as 95%, power as 80% the estimated sample size was 78. Adding 10% as the attrition rate and rounding off, the total sample size came to 90. Hence 30 patients were included in each group.

The groups were as follows,

Group A : Age and sex matched control group which consisted of patients who were attending the Medicine OPD for illnesses other than cardiovascular diseases.

Group B : CKD patients of stage 1, 2 and 3.

Group C : CKD patients of stage 4 and 5 who were undergoing haemodialysis for more than 3 months.

Sampling strategy:

A convenient sampling strategy was adopted. All CKD patients without any prior history of CKD (stages 4 and 5) who were admitted in the nephrology unit for hemodialysis and those CKD patients who were not undergoing hemodialysis (stages 1,2 and 3) were included in the study.

Inclusion criteria for CKD patients on Hemodialysis:

Stage 4 and stage 5 of renal failure

Patients on haemodialysis for more than 3 months Patients

on maintenance haemodialysis twice\thrice a week

Age 18-60 years.

Both sexes included.

Patients with no previous medical history of cardiac disease

Patient willing to take part in the study.

Exclusion criteria for CKD patients on Hemodialysis:

Patient not willing to take part in the study.

Age less than 18 years and more than 60 years

Patients with previous medical history and treatment of cardiac disease Patients

irregular on haemodialysis.

Inclusion criteria for CKD patients not on Hemodialysis:

Stage 1,2 and 3 of renal failure Age

18-60 years.

Both sexes included.

Patient willing to take part in the study.

Exclusion criteria for CKD patients not on Hemodialysis:

Patient not willing to take part in the study. Age less

than 18 years and more than 60 years **Inclusion**

criteria for control group:

Age 18-60 years.

Both sexes included.

Patients attending medicine OPD
Patients with no previous medical history of kidney disease

Patient willing to take part in the study **Exclusion**

criteria for control group:

Patient not willing to take part in the study

Age less than 18 years and more than 60 years

Patients with previous medical history and treatment of kidney disease **Study**

procedure:

Collection of blood samples

As per protocol, 5ml of blood was collected in serum separator red vacutainer from cubital vein under aseptic precaution. In laboratory blood was allowed to clot and centrifuged at the speed of 3000 revolution for 5 minutes. Serum was analysed for CK-MB and Troponin I by chemiluminescence immunoassay.

ESTIMATION OF CK-MB

Principle: Using the Access Immunoassay Systems, the Access CK-MB assay is a paramagnetic particle, chemiluminescent immunoassay for the quantitative measurement of CK-MB levels in human serum and plasma.

CKMB assay is a two-site sandwich immunoassay using direct chemiluminometric technology, which uses constant amounts of two antibodies. The first antibody, in the Lite Reagent, is a monoclonal mouse anti-CK-MB antibody labeled with acridinium ester. The second antibody, in the Solid Phase, is a monoclonal mouse anti-CK-BB antibody, which is covalently coupled to paramagnetic particles.

After incubation in a reaction vessel, materials bound to the solid phase are held in a magnetic field while unbound materials are washed away. Then, the chemiluminescent substrate is added to the vessel and light generated by the reaction is measured with a luminometer. The light production is directly proportional to the concentration of CK-MB in the sample. The amount of analyte in the sample is determined from a stored, multi-point calibration curve.

Sample Volume:

This assay requires 100 µL of sample for a single determination. This volume does not include the unusable volume in the sample container or the additional volume required when performing duplicates or other tests on the same sample.

REAGENTS

Well	Ingredients
R1a	Paramagnetic particles coated with anti-biotin antibodies
R1b	Purified mouse IgG and purified IgG buffered solution with BSA
R1c	Monoclonal anti-human CK MB antibody alkaline phosphatase

REFERENCE RANGE

Sample type	Median age	Age range	Reference interval
Plasma and serum	48	23-78	0.6-6.3 ng/mL

ESTIMATION OF TROPONIN I

PRINCIPLE

The Access high sensitivity troponin assay (hsTnI) is a paramagnetic particle, chemiluminescent immunoassay for high sensitivity quantitative determination of cardiac troponin I (cTnI) levels in human serum and plasma using the Access Immunoassay Systems to aid in the diagnosis of myocardial infarction (MI).

METHODOLOGY

The Access hsTnI assay is a sequential two–step immunoenzymatic (“sandwich”) assay. Monoclonal anti–cTnI antibody conjugated to alkaline phosphatase is added to a reaction vessel along with a surfactant–containing buffer and sample. After a short incubation, paramagnetic particles coated with monoclonal anti–cTnI antibody are added. The human cTnI binds to the anti–cTnI antibody on the solid phase, while the anti–cTnI antibody–alkaline phosphatase conjugate reacts with different antigenic sites on the cTnI molecules. After incubation in a reaction vessel, materials

bound to the solid phase are held in a magnetic field while unbound materials are washed away. Then, the chemiluminescent substrate is added to the vessel and light generated by the reaction is measured with a luminometer. The light production is directly proportional to the concentration of cTnI in the sample.

REAGENTS

Well	Ingredients
R1a	Dynabeads paramagnetic particles coated with monoclonal anti-human cTnI antibody
R1b	0.1 NaOH
R1c	TRIS buffered saline, surfactant, protein
R1d	Sheep monoclonal anti-human cTnI alkaline phosphate

Sample Volume

This assay requires 100 µL of sample for a single determination.

Reference range:

Sample type	Median age	Age range	Reference interval
Plasma and serum	48	23-78	24-30 pg/mL

ESTIMATION OF CREATININE Method:

Modified Jaffe's Method **Principle:**

Creatinine forms an orange colored complex with picric acid in an alkaline medium. The intensity of colour formed within fixed time directly proportional to the amount of creatinine present in the sample.

Reference values : Men : 0.6 - 1.2 mg/dl Female : 0.5-0.9 mg/dl

ESTIMATION OF UREA

Method: UV-GLDH method

Principle:

urease

Urea +H2O ⟶ Ammonia +CO2

GLDH

Ammonia +KG+NADH ⟶ Glutamate +NAD

Reference values:

Serum Urea : 10-45 mg/dl

ESTIMATION OF TOTAL BILIRUBIN

Method: Dimethylsulphoxide (DMSO) method

Principle: The Azobilirubin produced by the reaction between bilirubin and the diazonium salt of sulphanilic acid shows a maximum absorption at 555nM in acid medium. The intensity of the colour produced is proportional to the quality of bilirubin which has reacted. In the absence of an accelerator, only conjugated bilirubin reacts.in the presence of an accelerator, dimethylsulphoxide(DMSO) non-conjugated bilirubin also participates in the reaction.

Reference values: Up to 1.0 mg/dl

ESTIMATION OF SGPT:

Method: Modified IFCC

Principle: L-Alanine and alpha -ketoglutarate react with presence of Glutamic - Pyruvic Transaminase (GPT) in sample to yield pyruvate and L-Glutamate. Pyruvate is reduced by lactate dehydrogenase to yield lactate with the oxidation of NADH to NAD.The reaction is monitored by measurement of decrease in absorbance of NADH at 340 nm. The rate of reduction in absorbance is proportional to SGPT activity in sample.

GPT

L-Alanine+ Alpha - ⟶ Ketoglutarate Pyruvate +

L-Glutamate Pyruvate + NADH ⟶ (LDH) Lactate + NAD

Reference values: **5-40 IU/L**

ESTIMATION OF SGOT:

Method: Modified IFCC

Principle: SGOT reacts with L-Aspartate and -Ketoglutarate to form Oxaloacetate& L-Glutamate. The oxaloacetate reacts with NADH in presence of Malate Dehydrogenase (MDH) to form NAD.The rate of absorbance is directly proportional toSGOT activity in sample.

$$\text{Oxaloaceate + NADH} \xrightarrow{\text{MDH}} \text{Malate +NAD}$$

Reference values: 0-40 IU/L

ESTIMATION OF TOTAL PROTEIN:

Method: Biuret

Principle: In presence of alkaline cupric sulphate, the proteins produce violet colour. Intensity of colour is proportional to protein concentration.

Reference values: 6-8 gm/dl

ESTIMATION OF ALBUMIN:

Method: BCG

Principle: At 4.2 pH, bromocresol green binds with albumin, producing blue green colour. Intensity of colour is proportional to albumin concentration.

$$\text{Albumin + BCG} \longrightarrow \text{Albumin-BCG complex (blue)}$$

Reference values: 3.5-5.0 gm/dl

ESTIMATION OF ALBUMIN -GLOBULIN RATIO:

Calculated using equation albumin/ (total protein-albumin)

Reference values: 1.2-2.5

DATA AND STATISTICAL ANALYSIS:

All the data collected from the participants was compiled in a Microsoft Office Excel Sheet and analyzed using R/Jamovi Software. The categorical data were represented as frequencies and percentages and the quantitative data were represented as mean and standard deviation. Results will be shown in a Tabular and Graphical format. Appropriate Statistical tests were applied where ever necessary.

Ethical consideration:

The study was conducted only after obtaining approval from the Institutional Ethics Committee (IEC/PG/20/Jan/2021). Informed written consent was obtained from the patients prior to the study.

Results:

The majority of the study participants (61.1%) belonged to the age group of 45 to 60 years followed by those belonging to the age group of 25 to 44 years (32.2%). Most of the study participants were males (61.1%). The male to female ratio was 1.6:1. (Table 1). Majority of the study participants (92.2%) did not have any family history of CKD. The median duration of CKD among the study participants in groups B and C was found to be 2.5 years and 1 year respectively. (Figure 1). The measurement of various laboratory parameters is shown in Table 2. There is a significant difference between the three groups in all of the laboratory parameters except for Serum Bilirubin, AST and ALT. CKMB and Troponin I were found to be higher in Group C. (Table 3).

Discussion:

CKD is a condition characterized by a gradual loss of kidney function over time. (10) Chronic kidney disease is common in 10-13 percent of the population. It is irreversible, progressive, and linked to an increased risk of cardiovascular disease. Patients with this disease are usually asymptomatic, only presenting with the difficulties associated with renal failure in later stages. It can be treated conservatively (in patients who don't need dialysis but have a glomerular filtration rate of more than 15 mL/min) or with replacement therapy(hemodialysis, peritoneal dialysis and kidney transplantation).(52)The mean age in the three groups and the relative distribution shows majority i.e., 55 (61.1%) participants were in the age group of 45-60 years followed by those in 25-44 years (32.2%). After age 40, every decade GFR decreases by 10 ml/min, until it has dropped by roughly 30 ml/min by the age of 70. Comorbidities and risk factors such as hypertension and diabetes are on the rise, predisposing this population to a high burden of CKD. Individuals older than 65 years without hypertension or diabetes also have creatinine levels that progress to stage 3, possibly due to intrinsic renal disease or the natural ageing process. (11) Men are more prone than women to develop renal failure, hence they are a risk factor to predict kidney failure. Men may be at increased risk of kidney failure sooner than women because of differences in hormone levels. Higher testosterone levels in men

may cause a loss in kidney function. Men's kidney function may diminish faster than women's due to the protective benefits of oestrogens in women and/or the harmful effects of testosterone, as well as unhealthy lifestyles (12). Majority of patients I. e, 83 (92.2%) did not have any family history of CKD in our study. But Incidence of individuals with ESRD frequently have a family history of ESRD. It's linked to family history of diabetes, hypertension, anaemia, the metabolic syndrome, and socioeconomic status.

Study by Barry I Freedman et al showed approximately 23% of dialysis patients have close relatives with history of ESRD.(13). In study patients with and without ESRD, serum AST and ALT levels were considerably higher than in controls. The values, however, did not differ much between patients with and without ESRD. Serum aminotransferase levels in CKD patients on HD are lower. The causes of this decrease are unknown; however, they are likely to begin before to dialysis and are owing in part to hemodilution in patients prior to dialysis. According to study by Rasalkar and Nagalraj in CKD patients, mean creatinine value is 6.3±1.6 and serum urea 74.38±17.79 (14)Roxana Chicas,Jacqueline Mix et al on review of literature stated that CKD of undetermined etiology is multifactorial disease and have characteristics of increased Serum Creatinine, low GFR ,electrolyte abnormalities even in asymptomatic patients.(15) Luis Henrique Bezera et al on study on Hemodialysis patients with chronic renal disease showed lower serum levels of aminotransferases due to haemodilution.(16).Study by Isabella Ramios De Oliveria et al showed the levels of aminotransferases that were collected before the hemodialysis session were significantly lower than the values collected after the session.(17)

Cardiovascular troponins have largely replaced CK-MB as a diagnostic and prognostic biomarker of myocardial injury. The determination of serum CK-MB has demonstrated to be effective in the accurate diagnosis of cardiac damage, particularly acute coronary syndromes, in the majority of cases. Measurement of cardiac troponins, especially with high-sensitivity assays, could become a routine approach for stratifying cardiovascular risk and detecting preclinical CVD in patients with CKD. Increased cTn& CK-MB has been linked to left ventricular hypertrophy and possibly coronary disease in people with ESRD.

Corte Z, García C et al study showed, observed cTnI values of 98 percent of ESRD patients are above the reference range criterion. (18)

Conclusion:

There is definitive increase of CKMB and troponin I values in patients with chronic kidney failure on maintenance haemodialysis without coronary complications as compared to chronic kidney disease patients without haemodialysis without coronary complications

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As a result, we can infer that CKMB-mass and Troponin I testing can be used as a marker for all patients undergoing hemodialysis for CKD without coronary complications for earlier detection of CVD

Larger sample sizes, on the other hand, can help to clarify progression of CVD in CKD patients on hemodialysis.

Declarations:

a. Ethics approval and consent to participate:

The study was conducted only after obtaining approval from the Institutional Ethics Committee (IEC/PG/20/Jan/2021). Informed written consent was obtained from the patients prior to the study. (File attached)

b. Consent for publication

The names of the participants have not been included in the data sheet. Only the identification number has been included. Informed consent has been sought from the participants for publication of data.

c. Availability of data and materials

All data generated or analysed during this study are included in this published article [and its supplementary information files]

d. Funding - NA

This is a totally unfunded research project. NA

e. Authors' contributions

A.N and L.A – Generated and conceptualized the research topic. Helped with protocol writing, data collection and writing the results and discussion part of the research.

A.N, N.P and S.K – formulated the analysis plan, analysed the data and provided results in a presentable format.

References:

1. Jameson. disorders of the kidney and urinary tract. In: harrison's principles of internal medicine Harrison's principles of internal medicine. 20th ed. p. 2111. No Title. In.
2. Kidney Anatomy: Overview, Gross Anatomy, Microscopic Anatomy [Internet]. [cited 2022 Jan 10].Available from: https://emedicine.medscape.com/article/1948775-overview#a7
3. Chronic Kidney Disease (CKD): Practice Essentials, Pathophysiology, Etiology [Internet]. [cited 2022 Jan 10]. Available from: https://emedicine.medscape.com/article/238798-overview#a3
4. Berns JS. Routine screening for CKD should be done in asymptomatic adults... selectively. Clinical journal of the American Society of Nephrology : CJASN

[Internet]. 2014 [cited 2022 Jan 10];9(11):1988–92. Available from: https://pubmed.ncbi.nlm.nih.gov/25237073/

5. Varughese S, Abraham G. Chronic Kidney Disease in India. Clinical Journal of the American Society of Nephrology [Internet]. 2018 May 7 [cited 2022 Jan 10];13(5):802–4.Available from: https://cjasn.asnjournals.org/content/13/5/802
6. Dialysis Complications of Chronic Renal Failure: Practice Essentials, Electrolyte Abnormalities, Neurologic Complications [Internet]. [cited 2022 Jan 10]. Available from: https://emedicine.medscape.com/article/1918879-overview
7. A Personalized Approach to CKD and Cardiovascular Disease [Internet]. [cited 2022 Jan 10]. Available from: https://www.medscape.com/viewarticle/951859_5
8. Freda BJ, Tang WHW, van Lente F, Peacock WF, Francis GS. Cardiac troponins in renal insufficiency: review and clinical implications. Journal of the American College of Cardiology [Internet]. 2002 Dec 18 [cited 2022 Jan 17];40(12):2065– 71.Available from: https://pubmed.ncbi.nlm.nih.gov/12505215/
9. Chronic kidney disease (CKD) - Symptoms, causes, treatment | National Kidney Foundation [Internet]. [cited 2022 Jan 10].
10. Ammirati AL. Chronic Kidney Disease. Revista da Associacao Medica Brasileira (1992) [Internet]. 2020 [cited 2022 Feb 8];66Suppl 1(Suppl 1):3–9. Available from: https://pubmed.ncbi.nlm.nih.gov/31939529/
11. Gonçalves FA, Dalosso IF, Borba JMC, Bucaneve J, Valerio NMP, Okamoto CT, et al. Quality of life in chronic renal patients on hemodialysis or peritoneal dialysis: a comparative study in a referral service of Curitiba - PR. Jornal brasileiro de nefrologia : 'orgao oficial de Sociedades Brasileira e Latino- Americana de Nefrologia [Internet]. 2015 Oct 1 [cited 2022 Feb 9];37(4):467–74.Available from: https://pubmed.ncbi.nlm.nih.gov/26648496/

12. Brar A, Markell M. Impact of gender and gender disparities in patients with kidney disease. Current opinion in nephrology and hypertension [Internet]. 2019 Mar 1 [cited 2022 Mar 6];28(2):178–82.

13. Freedman BI, Volkova N v., Satko SG, Krisher J, Jurkovitz C, Soucie JM, et al. Population-based screening for family history of end-stage renal disease among incident dialysis patients. American journal of nephrology [Internet]. 2005 Nov [cited 2022 Mar 6];25(6):529–35.

14. G C, Rasalkar K, Naga Raju K. Levels of creatine kinase MB-mass in chronic kidney disease patients on maintenance hemodialysis without coronary complications. International Journal of Clinical Biochemistry and Research. 2019 Jun 28;6(2):161–4.

15. Chicas R, Mix J, Mac V, Flocks J, Dickman NE, Hertzberg V, et al. Chronic Kidney Disease Among Workers: A Review of the Literature. Workplace health & safety [Internet]. 2019 Sep 1 [cited 2022 Mar 6];67(9):481–90. Available from: https://pubmed.ncbi.nlm.nih.gov/31179873/

16. Cavalcanti Sette LHB, Lopes EP de A. Liver enzymes serum levels in patients with chronic kidney disease on hemodialysis: a comprehensive review. Clinics (Sao Paulo, Brazil) [Internet]. 2014 [cited 2022 Mar 6];69(4):271–8.

17. de Oliveira Liberato IR, de Almeida Lopes EP, de Mattos Cavalcante MAG, Pinto TC, Moura IF, Loureiro L. Liver enzymes in patients with chronic kidney disease undergoing peritoneal dialysis and hemodialysis. Clinics (Sao Paulo, Brazil)

[Internet]. 2012 [cited 2022 Mar 6];67(2):131–4. Available from: https://pubmed.ncbi.nlm.nih.gov/22358237/

18. Corte Z, García C, Venta R. Biological variation of cardiac troponin T in patients with end-stage renal disease and in healthy individuals. Annals of clinical biochemistry [Internet]. 2015 Jan 19 [cited 2022 Feb 28];52(Pt 1):53–60. Available from: https://pubmed.ncbi.nlm.nih.gov/25005457/

INFORMED CONSENT FORM (ENGLISH)

1. I, Mr./ Mrs ……., age……years residing at……. Hereby give my informed consent to participate in the study titled "A STUDY TO ESTIMATE THE LEVELS OF CREATINE KINASE-MB (CK-MB) AND TROPONIN I IN CHRONIC KIDNEY DISEASE PATIENTS ON HAEMODIALYSIS WITHOUT CORONARY ARTERY DISEASE"
2. There is no compulsion on me to participate in this study and I am giving my free consent for it.
3. I am ready and willing to undergo all tests and treatments in the present study.
4. I have been informed about investigations and/or treatment required for the study, no expenses will be borne by me for investigations and/or treatment which is related to this study. 5. I have read and I have been explained the general information and purpose of the present study.

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6. I have been informed/I have read the probable complications while participating in the present study.
7. I know that I can withdraw from the present study at any time.
8. Any data or analysis of this study will be purely used for scientific purpose and my name will be kept confidential except when required for any legal purpose.

Date:

Name and Signature of the study participant:

Thumb Print: