COMPARITIVE STUDY TO EVAULATE THE SAFETY AND EFFICACY OF FUROSEMIDE Vs CALCIUM POLYSTYRENE SULFONATE IN MANAGING HYPERKALEMIA

PROTOCOL

Submitted to DDH Ethics Committee

For the approval of protocol for the award of the degree of DOCTOR OF PHARMACY under JNTUH

SUBMITTED BY

Syed Omer Ahmed	20CE1T0024
T.S.V. Sravya	20CE1T0026
T. Manisha	20CE1T0027

UNDER THE ESTEEMED GUIDANCE OF

INSTITUTIONAL GUIDE	HOSPITAL GUIDE
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DEPARTMENT OF DOCTOR OF PHARMACY, BHARAT SCHOOL OF PHARMACY

Mangalpally, Hyderabad, 501510; 2023-2024

DECLARATION

My-self/we, Syed Omer Ahmed (20CE1T0024) ,T.S.V. Sravya (20CE1T0026) & T.Manisha(20CE1T0027) students of PHARM D (Doctor of pharmacy) 5th year, hereby declare that the project work entitles **"COMPARITIVE STUDY TO EVALUATE THE SAFETY AND EFFICACY OF FUROSEMIDE Vs CALCIUM POLYSTYRENE SULFONATE IN MANAGING HYPERKALEMIA "** is an original project work which will be carried out in the department of NEPHROLOGY, Durgabai Deshmukh Hospital and Research Centre for the fulfilment of award of degree of Doctor of Pharmacy under the supervision of institutional guide.

DATE:

NAME OF THE CANDIDATE:

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INDEX

ABBREVATIONS

HK	HYPERKALEMIA
CKD	CHRONIC KIDNEY DISEASE
AKI	ACUTE KIDNEY INJURY
GFR	GLOMERULAR FILTRATION RATE
K	SERUM POTASSIUM
Na	SODIUM
Ca	CALCIUM
P	PHOSPHOROUS

INTRODUCTION

HYPERKALEMIA: The most frequent electrolyte imbalance seen in kidney disease patients is hyperkalemia, especially in individuals who also have diabetes, heart failure, or are taking rennin angiotensin aldosterone system inhibitors.[1]

- Hyperkalemia is a dangerous medical condition that frequently shows up in individuals with heart failure and chronic kidney disease.
- Although they are known to enhance the course of various disease condition, RAAS inhibitors can also result in drug induced hyperkalemia.[2]
- Hyperkalemia is linked to arrhythmias, weakness, paralysis, and higher death rates
- In severe stages of chronic kidney disease, one of the most common reasons to start renal replacement therapy right once is greater serum potassium levels that are resistant to treatment.[3]
- The most serious consequences of hyperkalemia is a variety of cardiac dysrhythmias that can cause cardiac arrest and even death.[4]
- The primary cause of the rise in serum potassium over the normal range is low glomerular filtration rate.[5]
- The transmembrane potential of the excitable membrane found in nerve and muscle cells is mostly determined by potassium.[6]
- The first line treatment of protecting the heart and kidneys is RAAS blockers, however because of potassium rise, their usage is frequently restricted, leading to high rates of dropout.[7]

TYPES OF HYPERKALEMIA:

- Mild : 5.5 – 6.5meq/L
- Moderate : 6.5 – 7.5meq/L
- Severe : >7.5meq/L

CAUSES:

- Impaired excretion
- Increased intake [potassium supplements]
- Pseudohyperkalemia
- Medication use also [8]

EPIDEMOLOGY:

- The overall prevalence of hyperkalemia (K>5.5mmol/l) in whole population analysed was 12.6%
- The prevalence was 9.6% in CKD group
- 16.4% in HD group
- 10.6% in the CAPD group
- In terms of severity, the prevalence of hyperkalemia in population analyzed was over 7.5%
- For, Mild 3.2%
- Moderate 1.0%
- Severe 0.9%
- The connection between hyperkalemia and all causes of death results that individual with potassium readings above 6.0mmol/l had an increased mortality risk within the chronic kidney disease group.
- The study conducted by the latts et al evaluated the prevalence of hyperkalemia in almost 1.7 million patients with concurrent heart failure and stages 3 & 4 of chronic renal disease.[9]

SIGNS & SYMPTOMS:

- Heart palpitations
- Muscle weakness
- Nausea and vomiting
- Chest pain
- Diarrhea
- Arrhythmia

PATHOPHYSIOLOGY:

- Decreased renal excretion or extracellular potassium changes are the two main pathophysiologies of hyperkalemia.
- Common causes of hyperkalemia include improper potassium distribution, reduced renal excretion and pseudohyperkalemia. unless underlying disease is present, increased dietary potassium intake or other exogenous sources rarely induce more than temporary hyperkalemia states. Chronic hyperkalemia is variably linked to renal potassium excretion.

1.DECREASED RENAL EXCRETION:

The distal parts of the nephron are primarily responsible for the majority of potassium excretion, and the kidney plays a crucial role in maintaining appropriate potassium homeostasis. Renal disorders that result in hyperkalemia include:

- Renal tubular secretory abnormalities
- Impaired renin aldosterone axis
- Drug induced hyperkalemia

RENAL TUBULAR SECRETARY ABNORMALITIES:

Prevalent anomalies in renal tubular secretory function that may result in renal illness in sickle cell disease, renal tubular acidosis type1 and hyperkalemia.

IMPAIRED RENIN ALDOSTERONE AXIS:

Adrenal enzyme deficits and medication can affect the rennin-aldosterone axis, which can lead to exacerbated hyperkalemia in Addison's disease**.**

DRUG INDUCED HYPERKALEMIA:

Medication can cause hyperkalemia through many mechanisms than interfering with the rennin-aldosterone axis. Potassium sparing diuretics prevent sodium reabsorption in the distal nephron, which lowers the luminal voltage gradient and decreases of potassium excretion rates.

2. DECREASED DISTAL TUBULAR FLOW WITH LOW SODIUM:

Hyperkalemia is also brought on by a substantial reduction in tubular flow rate or sodium supply at the distal nephron.

3.RENAL FAILURE:

The most common causes of oliguric acute kidney failure are intestinal nephritis and acute tubular necrosis. Because of frequent injury, the collecting duct cells and distal tubules are unable to discharge potassium furthermore, as previously mentioned, distal tubular flow rate or sodium supply are frequently reduced, which results in hyperkalemia.

4.ABNORMAL POTASSIUM DISTRIBUTION:

When metabolic acidosis, insulin insufficiency, aldosterone deficit and tissue injury occur, anomalies in potassium distribution are observed. During metabolic acidosis, there is a large extracellular or intracellular shift of potassium in exchange for protons, leading to hyperkalemia.[10]

DIAGNOSIS

- Clinical history
- Physical examination
- Review of medications
- Assessment of cardiac function, kidney& urinary tract
- Assessment of hydration status
- Electrocardiogram
- Comprehensive laboratory workup[11]

TREATMENT

The treatment strategies of hyperkalemia may focus on these several targets from a pathophysiological perspective.

- Stabilization of cell membrane potential
- Transferring of potassium from extracellular gaps into cells, reducing potassium concentrations,
- Improving potassium excretion by intravenous calcium.

Using insulin, glucose, or beta -adrenergic agonists can stimulate potassium redistribution, these therapies are frequently chosen in emergency interventions since they rapidly lower potassium levels in a matter of minutes.

Lowering the body's potassium content is necessary to restore potassium balance.

This can be accomplished by reducing the amount of potassium that is consumed, using drugs such as cation-Exchange resins or loop diuretics that enhance potassium excretion through the gastrointestinal tract, urine or alternatively, by using hemodialysis, which lowers the potassium level of the body.

Among the several options for treating hyperkalemia, GI potassium elimination has received more attention lately mostly due to the availability of novel medications such sodium zirconium cyclosilicate and patriomer.

Historically, the old cation exchange resin sodium polystyrene sulfonate [SPS] and its derivative calcium polystyrene sulfonate had been the only alternatives available for encouraging potassium removal via the GI.

MANAGEMENT APPROACHES TO HYPERKALEMIA

ACUTE MANAGEMENT:

Prevent Neuromuscular abnormalities and potentially fatal cardiac conduction

Transfer potassium into the cell

Get rid of extra potassium

STABILIZE: Evaluate patient for life threatening toxicities

- Calcium gluconate
- Initiate ECG monitoring

SHIFT: Augment the shift of potassium from extra to intracellular space

- Dextrose infusion
- Beta adrenergic agonist
- Sodium bicarbonate

REMOVE: Renal replacement therapy

- Hemodialysis
- Potassium binders

CHRONIC MANAGEMENT:

Prevent hyperkalemia from developing or from returning.

Fix any underlying issues with potassium homeostasis

Enhance the excretion of potassium

REMOVE: Manage any ongoing contributor to hyperkalemia

- Diet
- Medication
- Comorbid condition

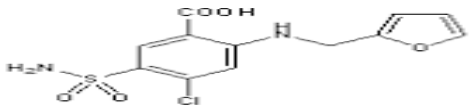
INCREASED EXCRETION OF POTASSIUM:

- Furosemide [loop diuretics]
- Mineral corticoid
- Potassium binders [SPS, CPS][12]

DRUGS IN THE STUDY

1.FUROSEMIDE (LOOP DIURETICS)

CHEMICAL STRUCTURE:



Molecular formula: $C_{12}H_{11}ClN_2O_5S$

Trade names: Furocot, Lasix

MOA : It mainly works by inhibiting electrolyte reabsorption from the kidneys and enhancing the excretion of water from the body. It acts by inhibiting the luminal Na-K-cl co transporter in the thick ascending limb of loop of henle,by binding to Na-K-2cl transporter, that causing more sodium, chloride and potassium to excreted in the urine.

Dosing forms available: oral: 20-80mg OD

IV/IM: 20-40mg

Dose: HYPERKALEMIA (0.5 – 1mg/kg/day)

ANTI HYPERTENSIVE (20-80mg/kg PO/BID)

Pharmacokinetics:

Route – oral, IV, IM

Bioavailability – 47-64%

Protein binding – 91-99%

Onset – 30-60min(oral)

30mins (IM)

5mins (IV)

Duration- 6-8hrs (oral)

2hrs (IV)

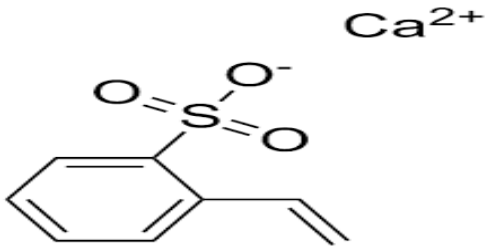
Metabolism -liver (Glucordination)

Half life - 30-120mins

Excretion – urine (2ml/min/kg) [13]

2.CALCIUM POLYSTYRENE SULFONATE

CHEMICAL STRUCTURE:



Molecular formula – $(C_8H_8O_3S)x.xCa$

Trade names – Kayexalate, kionex, Kalimate

MOA – Polystyrene sulfonate which is not absorbed binds excess potassium ,carrying it out of the body. The indigestible potassium polystyrene sulfonate complex is excreted with the faeces , preventing the absorption of potassium into the bloodstream. Hence, the serum potassium level decreases.

Dosing forms available – oral – 15g (3-4 times daily)

Rectal – 30g in 150ml of water

10% dextrose in water

Pharmacokinetics- Route- oral, rectal

Distribution- it is not absorbed, the distribution of resin limited to GIT

Metabolism- it is metabolized by glucuronidation. During it pass through the colon , it exchanges the majority of its sodium ions for potassium ions, where as resin remain inalterable.

Excretion- Faecus

Halflife- 8-16hrs.[14]

LITERATURE REVIEW

1.F.John Gennari, MD

Disorders of potassium homeostasis (Hypokalemia and hyperkalemia) 2001 april 2nd USA ,(CCC)

Hypo-and hyperkalemia are the most commonly encountered electrolyte abnormalities in hospitalized patients . Although no data are available concerning their specific prevalence in critically ill patients, one might expect these abnormalities to occur frequently because of the disruption in normal homeostasis inherent in critical illness and the multiple interventions necessary for care. Hypo-and hyperkalemia are more likely to contribute to morbidity and mortality in this group of patients.

2.Yelena Mushiyakh, MD,Harsh Dangaria, MD,Shahbaz Qavi, MD,Noorjahan Ali, MD,John Pannone , MD.

Treatment and pathogenesis of acute hyperkalemia,2012 JAN 26th,USA(PMC)

This article focuses on the pathogenesis, clinical manifestations, and various treatment modalities for acute hyperkalemia and presents a systematic approach to selecting a treatment strategy. This article presented guidelines to aid clinicians in their diagnosis and treatment of this potentially life-threatening condition.

3.ANTHONY J. VIERA, MD, MPH, AND NOAH WOUK, MD

Potassium Disorders: Hypokalemia and Hyperkalemia 2015, sep 15 (AFP)

Hypokalemia and hyperkalemia are common electrolyte disorders caused by changes in potassium intake, altered excretion, or transcellular shifts. For both disorders, it is important to consider potential causes of transcellular shifts because patients are at increased risk of rebound potassium disturbances.

4. Mario V Beccari and Calvin J Meaney

Clinical utility of patiromer, sodium zirconium cyclosilicate, and sodium polystyrene sulfonate for the treatment of hyperkalemia: an evidence-based review. 2017,march 23(PMID) USA

The objective of this article was to review the efficacy and safety evidence for patiromer, sodium zirconium cyclosilicate (ZS9), and sodium polystyrene sulfonate (SPS) for the treatment of hyperkalemia.Patiromer and ZS9 have improved upon the age-old standard SPS for the treatment of hyperkalemia.

5. Biff F. Palmer, MD and Deborah J. Clegg, PhD

Diagnosis and treatment of hyperkalemia.2017, DECEMBER 12(CCJM)

Excessive intake of potassium can cause hyperkalemia but usually in the setting of impaired renal function. We discuss the clinical manifestations of hyperkalemia and outline an approach to its diagnosis and treatment.

6.Mi-Yeon Yu, Jee Hyun Yeo, Joon-Sung Park, Chang Hwa Lee, and Gheun-Ho Kim*

Long-term efficacy of oral calcium polystyrene sulfonate for hyperkalemia in CKD patients 2017 MARCH 22nd, Japan(PMC)

Calcium polystyrene sulfonate (CPS) has long been used to treat hyperkalemia in patients with chronic kidney disease (CKD). However, its efficacy and safety profile have not been systematically explored. We investigated the long-term efficacy of oral CPS for treating mild hyperkalemia on an outpatient basis.

7. Toni De Stefano , Silvio Borrelli , Carlo Garofalo , Michele Provenzano , Luca De Nicola , Roberto Minutolo , Giuseppe Conte

Hyperkalemia treatment in chronic kidney disease patients: overview on new K binders and possible therapeutic approaches]2018,sep 5th Italian.(PMED)

However, few studies showed that SPS is efficacious to reduce serum K and is associated with increased risk of severe adverse effects. Furthermore, Patiromer, sodium free agent, might have a further advantage in CKD patients, reducing the salt intake in these patients. In addition, ZS-9, being fast-acting drug, might be used also in the treatment of acute hyperkalaemia.

8. Brit Long , Justin R Warix , Alex Koyfman

Controversies in Management of Hyperkalemia. 2018 MAY 3rd,Texas (pubmed)

This review evaluates the classic treatments of hyperkalemia and discusses controversies and new medications for management. Hyperkalemia can be deadly, and treatment requires specific measures including membrane stabilization, cellular shift, and excretion.

9. Stefano Bianchi,[1] Filippo Aucella,[2] Luca De Nicola,[3] Simonetta Genovesi,[4] Ernesto Paoletti,[5] and Giuseppe Regolisti[6]

Management of hyperkalemia in patients with kidney disease: a position paper endorsed by the Italian Society of Nephrology 2019,may 22(PMC)

HK is a common finding in patients with kidney disease, due to the effects of kidney dysfunction on potassium (K) homeostasis, and this condition strongly impacts upon the quality of life and prognosis of these patients.

The management of HK is a major yet unmet need in the Nephrology setting, in spite of the negative impact of this alteration on patient prognosis and the obvious high costs of hospitalization and dialysis

10. Lara Belmar Vega [1], Emilio Rodrigo Galabia [2], Jairo Bada da Silva [2], Marta Bentanachs González [2], Gema Fernández Fresnedo [2], Celestino Piñera Haces [2], Rosa Palomar Fontanet , Juan Carlos Ruiz San Millán [2], Ángel Luis Martín de Francisco 2019,MAY 17 ENGLAND(PUBMED)

Epidemiology of hyperkalemia in chronic kidney disease

To examine the prevalence of hyperkalaemia in CKD, identify factors associated with its appearance and the relationship between hyperkalaemia and mortality.

11. Biff F. Palmer, MD · Juan Jesus Carrero, PharmD, PhD· Deborah J. Clegg, PhD · Simon D. Roger, MD · Bruce S. Spinowitz, MD

Clinical Management of Hyperkalemia 2020,NOV 4th (MCP)

This review summarizes the physiology of hyperkalemia and suggests evidence-based clinical considerations that may provide improvements in care and outcomes in patients with an increased hyperkalemia risk.

12. Erasmia Sampani [1], Marieta Theodorakopoulou [1], Fotini Iatridi [1], Pantelis Sarafidis [1]

Hyperkalemia in chronic kidney disease: a focus on potassium lowering pharmacotherapy.2023 AUGUST,13th, Greece(pubmed)

Renin-angiotensin-aldosterone-system blockers are first-line treatments for cardio- and nephroprotection, but their use is often limited due to K^+ elevation, resulting in high rates of discontinuation.

AIM & OBJECTIVES

AIM : To determine the comparative study of safety and efficacy of furosemide versus calcium polystyrene sulfonate in treating hyperkalemia.

OBJECTIVES:

- To access the safety and efficacy of furosemide and calcium polystyrene sulfonate.
- To monitor the serum potassium levels in hyperkalemia condition.

METHODOLOGY

INSTITUTION OF STUDY: This study will be conducted in department of nephrology, Durgabai Deshmukh hospital and research centre , vidyanagar HYD.

STUDY DURATION: 6 months

STUDY DESIGN: Comparative and prospective study.

SAMPLE SIZE: 70

ETHICS STATEMENT: Study will be conducted only after the approval of hospital committee.

TOOL: Patients case reports, lab investigation reports.

STUDY CRITERIA

INCLUSION CRITERIA

- Hyperkalemia condition serum potassium level count above 6.0mmol/L
- Age group >18yrs
- Non dialysis patient
- Patient with AKI

EXCLUSION CRITERIA

- Pregnant and lactating women
- Pediatrics
- Patients who are on dialysis

PRIMARY ASSESSMENT

- To monitor the electrolyte levels
- To monitor the serum potassium levels

DATA COLLECTION

All the relevant and necessary data (demographic details, laboratory details and treatment) will be collected from the patient record and laboratory records.

STATISTICAL TOOL

The statistical tool includes z test, difference of mean

PROCEDURE

Hyperkalemia condition will be assessed in the study.

Base line demographic date will be collected from patient case reports.

Serum potassium levels are monitored of patient will be assessed in study.

The results obtained will be evaluated and analysed statistically.

PLAN OF WORK

Designing the patient data collection form

↓

Seeking permission from ethics committee

↓

All those patients who meet the study criteria will be included in the study after obtaining the informed consent form

↓

Assessment of patients with kidney failure

↓

Assessment of hyperkalemia

↓

The results will be analysed and evaluated statistically.

REFERENCES

(1). Management of hyperkalemia in patients with kidney disease: a position paper endorsed by the Italian Society of Nephrology

Stefano Bianchi,[1] Filippo Aucella,[2] Luca De Nicola,[3] Simonetta Genovesi,[4] Ernesto Paoletti,[5] and Giuseppe Regolisti[6] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6588653/

(2). Clinical utility of patiromer, sodium zirconium cyclosilicate, and sodium polystyrene sulfonate for the treatment of hyperkalemia: an evidence-based review

Mario V Beccari* and Calvin J Meaney

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5367739/

(3). **Hyperkalemia treatment in chronic kidney disease patients: overview on new K binders and possible therapeutic approaches]**

[Article in Italian]

Toni De Stefano 1, Silvio Borrelli 1, Carlo Garofalo 1, Michele Provenzano 1, Luca De Nicola 1, Roberto Minutolo 1, Giuseppe Conte 1

https://pubmed.ncbi.nlm.nih.gov/30234232/

(4). Controversies in Management of Hyperkalemia

Brit Long [1], Justin R Warix [2], Alex Koyfman [3]

https://pubmed.ncbi.nlm.nih.gov/29731287/

(5). Management of hyperkalaemia in chronic kidney disease

- Csaba P. Kovesdy

https://www.nature.com/articles/nrneph.2014.168

(6). Potassium Disorders:

Hypokalemia and Hyperkalemia

ANTHONY J. VIERA, MD, MPH, and NOAH WOUK, MD, University of North Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina

https://in.docs.wps.com/module/common/aiGuide?sa=wa&ps=1&fn=p487%20%281%29.pdf&sid=sIIyUqPn4AduWjrYG&v=v2#1724240839316

(7). Hyperkalemia in chronic kidney disease: a focus on potassium lowering pharmacotherapy

Erasmia Sampani [1], Marieta Theodorakopoulou [1], Fotini Iatridi [1], Pantelis Sarafidis [1]

https://pubmed.ncbi.nlm.nih.gov/37545002/

(8). Disorders of potassium homeostasis

Hypokalemia and hyperkalemia

- F.John Gennari, MD

https://www.criticalcare.theclinics.com/article/S0749-0704(01)00009-4/abstract

(9). Epidemiology of hyperkalemia in chronic kidney disease

[Article in English, Spanish]

Lara Belmar Vega [1], Emilio Rodrigo Galabia [2], Jairo Bada da Silva [2], Marta Bentanachs González [2], Gema Fernández Fresnedo [2], Celestino Piñera Haces [2], Rosa Palomar Fontanet [2], Juan Carlos Ruiz San Millán [2], Ángel Luis Martín de Francisco

https://pubmed.ncbi.nlm.nih.gov/30898450/

(10). Treatment and pathogenesis of acute hyperkalemia

Yelena Mushiyakh , MD,Harsh Dangaria , MD,Shahbaz Qavi , MD,

Noorjahan Ali , MD,John Pannone , MD &**David Tompkins**, MD

https://www.tandfonline.com/doi/full/10.3402/jchimp.v1i4.7372#d1e201

(11). Diagnosis and treatment of hyperkalemia

Biff F. Palmer, MD and Deborah J. Clegg, PhD

https://www.ccjm.org/content/84/12/934

(12). Clinical Management of Hyperkalemia

Biff F. Palmer, MD ,Juan Jesus Carrero, PharmD, PhD,Deborah J. Clegg, PhD,Simon D. Roger, MDBruce S. Spinowitz, MDMatthew R. Weir, MD

https://www.mayoclinicproceedings.org/article/S0025-6196(20)30618-2/fulltext

(13). Furosemide

Author links open overlay panelLaura Carone BMedSci [a], Stephen G. Oxberry PhD [b], Robert Twycross DM, FRCP [c], Sarah Charlesworth BPharm (Hons), DipClinPharm, MRPharmS [c], Mary Mihalyo BS, PharmD, RPh, CGP, BCPS, CDE [d], Andrew Wilcock DM, FRCP

https://www.sciencedirect.com/science/article/pii/S0885392416301191

(14). Long-term efficacy of oral calcium polystyrene sulfonate for hyperkalemia in CKD patients

Mi-Yeon Yu, Jee Hyun Yeo, Joon-Sung Park, Chang Hwa Lee, and Gheun-Ho Kim*

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5362098/

(15).K.Visweswara rao:BIOSTATISTICS A manual of statistical methods for use in health , nutrition and anthropology.

(16). K. Visewsara rao: BIOSTATISTICS IN BRIEF MADE EASY

DATA COLLECTION FORM

NAME:	AGE:	GENDER:
PATIENT ID NO:	HEIGHT:	WEIGHT:
PRESENT HISTORY:		
PAST MEDICATION HISTORY:		
PROVISIONAL DIAGNONIS:		
VITALS: **BLOOD PRESSURE:** **PULSE RATE:** **TEMPERATURE:** **RESPIRATORY:** **SPO 2:**		

PATIENT DETAILS

INVESTIGATIONS

ELECTROLYTE TEST	NORMAL RANGE	DAY 1	DAY2	DAY 3
SODIUM				
POTASSIUM				
CHLORIDE				
BICARBONATES				
CALCIUM				
PHOSPHOROPUS				

RENAL FUNCTION TEST

PARAMETER	DAY 1	DAY 2	DAY 3	DAY 4
GFR(ml/min)				
Urea(mg/dL)				
Creatinine				

TREATMENT

BRAND NAME	GENERIC NAME	DOSE

INFORMED CONSENT FORM

Title of the project:

COMPARATIVE STUDY TO EVALUATE THE SAFETY AND EFFICACY OF FUROSEMIDE AND CALCIUM POLYSTYRENE SULPHONATE IN MANAGEMENT OF HYPERKALEMIA.

Name of the participant:

Name of the principal investigator:

Name of the institution:

Documentation of informed consent:

I, …….. Have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over the age of ….. Years and exercising my free power of choice hereby give my consent to be included as a participant in this study.

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study
4. My rights and responsibilities have been explained to me by the investigator.
5. I have been advised that no risks are associated with my participation in the study.
6. I have informed the investigator of all the treatments I am taking or have taken in the past including the desi (alternative) treatments.
7. I agree to cooperate with the investigator and I will inform him/her immediately f I suffer any unusual symptoms.
8. I have not participated in any research study in the past.
9. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in the hospital.
10. I am also aware that the investigators may terminate my participation in the study at any time for any reason, without my consent.
11. I hereby give permission to the investigators to release the information obtained from me as a result of participation in this study to the regulatory authorities and ethics committee. I understand that they may inspect my original records.
12. My identity will be kept confidential if my data is publicly presented

13. I have had my questions answered to my satisfaction

14. I have decided to be in the study.

I am aware, that if I have any questions during this study, I should contact at once of the addresses listed above. By signing this consent form , I attest that the given information in this document has been clearly explained to me and apparently understood by me . I will be give a copy of this consent document.

Name and signature/thumb impression of the participant (or legal representative if participant is incompetent)

_____ (NAME)

Signature date: _____ Time:

Name and signature of impartial witness (required for illiterate patients)

_____ (NAME)

Signature date: _____Time:

Address and contact number of the impartial witness

Name of the investigator:

Address:

Contact number:

Signature of the investigator:

INVESTIGATER CERTIFICATE

I certify that all the elements including the nature, purpose and possible risks of the above study as described in the consent document have been fully explained to the subject. In my judgment, the participant possesses the legal capacity to give consent to participate in the research and is voluntarily and knowingly giving the consent to participate.

NAME OF STUDENT	REGISTRATION NO.	SIGNATURE
SYED OMER AHMED	20CE1T0024	
T.S.V. SRAVYA	20CE1T0026	
T. MANISHA	20CE1T0027	

HOSPITAL GUIDE	DESIGNATION	SIGNATURE
DR.PRAVEEN KUMAR ETTA (Consultant nephrologist)	MBBS,MD,DM	

INSTITUTIONAL GUIDE	DESIGNATION	SIGNATURE
DR.B.SWATHI	ASS.PROFESSOR (DEPT.PHARMACY PRACTICE)	

REMARKS OF SCIENTIFIC COMMITTEE

INVESTIGATER CERTIFICATE

I certify that all the elements including the nature, purpose and possible risks of the above study as described in the consent document have been fully explained to the subject. In my judgment, the participant possesses the legal capacity to give consent to participate in the research and is voluntarily and knowingly giving the consent to participate.

Signature of the investigator: Date:

Name of the investigator:

Address:

Contact number:

Signature of the investigator:

అనుమతి పత్రము

ప్రధాన దర్యాప్తుదారు పేరు :

సంస్థ పేరు : డిడిహెచ్ఆర్సీ, హైదరాబాద్

అధ్యయన శీర్షిక :

సమాచార పత్రము

ఈ పరిశోధనలో ..... లక్షణాలు యొక్క ప్రాబల్యం గురించి అధ్యయనం చేయుచున్నామని, ఇది కేవలం ఒక పరిశీలాత్మక అధ్యయనం మాత్రమేనని, ఈ పరిశోధనలో పాల్గొనే వ్యక్తి ఏ సమయంలోనైన వైదొలగే హక్కు ఉన్నదని పాల్గొన్న వ్యక్తికి వివరించడమైనది.

ఈ ప్రొటోకాల్ని మా సంస్థ ఎథికల్ కమిటీ ఆమోదించింది.

సమ్మతి ధృవీకరణ పత్రము

నేను ఈ సమాచారం చదవటం లేదా చదివించుకోవడమైనది. నేను దీని గురించి ప్రశ్నలు అడిగే అవకాశం నాకు కలిగినది. నేను అడిగిన ప్రశ్నలకు నా సంతృప్తి మేరకు జవాబు ఇచ్చినారు. ఈ పరిశోధనలో ఒక భాగస్వామిగా పాల్గొనేందుకు స్వచ్ఛందంగా సమ్మతించితిని మరియు నా వైద్య సంరక్షణ ప్రభావితం కాకుండా ఏ సమయంలోనైనా వైదొలగే హక్కు నాకు ఉన్నదని, నా వైద్య సంబంధ కార్యలు యాక్సెస్ చేసుకోవడానికి బాధ్యతాయుత వ్యక్తులచే పరిశీలించబడునని అర్థం చేసుకుని దీనికి నేను సమ్మతించితిని.

పాల్గొనే / పాల్గొన్న వ్యక్తి పేరు :

పాల్గొన్న వ్యక్తి సంతకం / వేలి ముద్ర :

తేది :

సాక్షి సంతకం :

తేది & సమయం

దర్యాప్తుదారుని సంతకం :

తేది & సమయం

అనుమతి పత్రము

1. నాకిచ్చిన ప్రత్యేక విషయాన్ని చదివి అర్థం చేసుకున్నాను.
2. నాకు వివరించిన సమాచార పత్రము నా దగ్గర వున్నది.
3. నాకు అధ్యయనం యొక్క విశేషతను గూర్చి వివరించారు
4. నా హక్కులు, బాధ్యతలను మా పరిశోధకుని ద్వారా తెలుసుకొన్నాను.
5. నేను/మేము ఈ అధ్యయనంలో పాల్గొనినట్లయితే ఎటువంటి కష్టాలు వుండవని నాకు/ మాకు తెలియ పరచారు.
6. ఏవైన అసహజ లక్షణాలతో బాధ పడినప్పుడు వాటిని మా పరిశోధకునికి తెలియపరచినప్పుడు, ఒప్పుకుంటు వారితో సహకరిస్తాము.
7. నేను/మేము ఏ సమయం లోనైనా ఈ చికిత్స నుండి బయటకు రావడం, ఆసుపత్రిలో జరిగే భవిష్య చికిత్స కు ఎటువంటి ఆటంకం కలిగించదు అని తెలుసుకొన్నాను
8. నేను/మేము పరిశోధకుని ఈ చికిత్స నుండి ఎప్పుడైనా ఎటువంటి కారణానైనా మా ప్రమేయం లేకుండా తొలగించ వచ్చని తెలుసుకొన్నాను.
9. నేను/మేము, పరిశోధకుని ద్వారా లభించిన విషయాన్ని పై ఎథికల్ కమిటీ ఆమోదించింది.
10. నా / మా బంధువు గురించి అందరి ముందు తెలియపరిచిన విషయాన్ని గుప్తంగా ఉంచబడుతుంది.
11. నాకు/ మాకున్న కొన్ని ప్రశ్నలకు వారి సమాధానలకు సంతృప్తి చెందాను.
12. మేము ఈ చికిత్సలో ఒక భాగమని నిర్ణయించుకొన్నము.

పాల్గొనే / పాల్గొన్న వ్యక్తి పేరు :

పాల్గొన్న వ్యక్తి సంతకం / వేలి ముద్ర :

తేది :

సాక్షి సంతకం :

తేది & సమయం :

దర్యాప్తుదారుని సంతకం :

తేది & సమయం

पेशेंट सहमति विधि

सहयोगी का नाम :

शोधकर्ता का नाम :

1.
2.

संस्था का नाम :

शोध कार्यपालक का नाम :

रोगी के लिए

मैं _____ मेरे चुनाव के मुफ्त अधिकार का उपयोग कर इस क्लिनिकल शोध में एक सदस्य बन्ने की सहमती देता हूँ!

मैं निम्नलिखित को मान्य करता हूँ

1. मेरी उम्र 18 साल से ज्यादा है!
2. इस शोध के दौरान मुझे कोई नई दावा नहीं दी जाएगी यह मैं जनता हूँ!
3. मैं यह भी जनता हूँ की इस शोध की जानकारी QOL के बेहतरी में मददगार होगी!
4. इस शोध के कारण एवं प्रक्रिया से मैं समाधाती हूँ, यह मैंने डॉक्टर से कबुल किया है!
5. डॉक्टर के द्वारा मुझे शोध के प्रक्रिया, अवधि एवं कारणों की एवं मुझे क्या करना है इस बात की सम्पूर्ण जानकारी दी गई है! मैंने जानकारी पात्र को पूरी तरह समझा है!

6. मुझे यह भी जानकारी है की मैं कभी भी इस शोध अभ्यास से अलग हो सकता हूँ! मुझे कोई स्पष्टीकरण देने की भी जरूरत नहीं है की मई मध्य में ही यह शोध क्यों छोड के जा रहा हूँ!

पेशेंट/सहयोगी का नाम एवं हस्ताक्षर, तिथि(date)

*गवाह/साक्षी का नाम एवं हस्ताक्षर, तिथि(date)

मैं यह पुष्टि करता हूँ के मैंने इस अभ्यास का स्वभाव एवं उद्देश्य _____

को सम्झादिया गया है!

शोधकर्ता के हस्ताक्षर

* साक्षी के हस्ताक्षर पेशेंट के अनपद होने के कारण ही ली जानी चाहिए! साक्षी यह सुनिश्चित करता है के पेशेंट को पेशेंट विजापना पत्र और पेशेंट सहमति विधि उसकी अपनी भाषा में सम्झादिया गया है!

साक्षी का नाम एवं पता :

_____

_____

_____