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RETROSPECTIVE COHORT STUDY OF DRUG UTILIZATION PATTERNS IN CHRONIC KIDNEY DISEASE (CKD) PATIENTS: A COMPARATIVE ANALYSIS OF DIFFERENT STAGES OF CHRONIC KIDNEY DISEASE

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ABSTRACT

Background: CKD is a progressive, irreversible condition characterised by the gradual decline in kidney function. CKD patients are frequently subjected to polypharmacotherapy for complications and comorbidities, resulting in a significantly high ADRs. It is necessary to achieve the optimal pharmacotherapy taking into account the patterns of drug utilization. **Aim:** To describe the use of drugs in CKD and study mechanisms of action of frequently prescribed agents at different stages of CKD using an in silico comparative analysis. **Methods:** Three frequently used drugs (Torsemide, Febuxostat and Furosemide) were studied. The structures of drugs were downloaded in SDF format from PubChem. ADMETlab 3.0 and Lipinski's Rule of Five were used to evaluate the pharmacokinetic properties and drug-likeness. The target proteins were predicted with Swiss Target Prediction, and the CKD-related genes were all obtained from GeneCards. Intersecting targets were then processed by the STRING and visualized in Cytoscape to identify hub genes. AutoDock Vina was employed for molecular docking and ShinyGO was used for function enrichment analysis. Structures were obtained from RCSB PDB, and pictures of drug-target interactions produced with PyMOL. **Results:** Network pharmacology analysis identified 205 shared targets and 10 key hub genes (SRC, HSP90AA1, BCL2, PPARG, PTGS2, GSK3B, BCL2L1, MAPK14, RHOA, and MTOR) linked to inflammation, oxidative stress, apoptosis, and metabolic regulation. Major enriched pathways included PI3K-Akt, cAMP, lipid and atherosclerosis, and nitrogen metabolism. Docking studies demonstrated strong binding affinities (−4.3 to −8.8 kcal/mol), with Torsemide showing the highest stability, followed by Febuxostat & Furosemide. **Conclusion:** The study highlights the importance of multi-target pharmacotherapy in CKD management, with Torsemide demonstrating superior therapeutic potential.

KEYWORDS: Chronic kidney disease, Drug utilization pattern, Prescribing trends, Retrospective cohort, Rational pharmacotherapy.

INTRODUCTION

Chronic kidney disease (CKD) is a horrific health issue in the world, which is marked by a progressive and irreparable impairment of the kidney function. It is stated that it is less effective in bicycle safety and security at the intersection.[1] CKD is a chronic structural or functional complication of kidney that persist more than three months and can have an adverse effect on general health.. In chronic kidney disease (CKD), there are five stages based on the estimated glomerular filtration rate (eGFR). End-stage renal disease (ESRD), which requires renal replacement therapy, is indicated by stage 5.[2] Chronic kidney disease (CKD) is predicted by the Global Burden of Disease (GBD) Study to rank among the top 10 major causes of mortality and be the fifth most common cause of death globally by 2040.[3] However, as kidney function deteriorates, managing medications for CKD becomes more complex due to shifts in pharmacokinetics and pharmacodynamics. In the advanced stages of chronic kidney disease (CKD), many drugs may need to be discontinued or their dosages adjusted to avoid toxicity resulting from reduced renal clearance (7; 15). To achieve optimal therapeutic outcomes, reduce adverse effects, and slow the progression of kidney damage, medication use in chronic kidney disease (CKD) must be approached with great caution. The evidence-based clinical practice guidelines, including those of the Kidney Disease: Improving Global Outcomes (KDIGO) suggest pharmacological interventions based on CKD stage and comorbidity-they can be phosphate binders, statins, erythropoiesis-stimulating agents, and renin-angiotensin system inhibitors.[9] Network pharmacology is a new field that combines systems biology and computational pharmacology and offers a framework to understand drug-target-pathway interactions holistically. It enables the visualization of complex biological networks, which aids researchers in identifying important molecular targets and signalling pathways that would be utilized to alter the illness.[12] Further, the study examines molecular pathways using in silico analysis to enhance the insight into the pharmacological effects of given medications and how they interact with the molecular targets in the context of chronic kidney disease. The results aim to enhance the use of medication rationally, enhance patient safety and make evidence-based decisions in the treatment in the management of chronic kidney disease.

MATERIALS AND METHODS

Definition of CKD

The KDIGO 2024 Clinical Practice Guidelines state that changes in kidney structure or function that last for at least three months and have an impact on health are indicative of chronic kidney disease. Decreased eGFR (<60 mL/min/1.73 m2) or evidence of kidney damage, including albuminuria, abnormalities in urine sediment, electrolyte imbalances, structural changes in the kidneys, or histological signs, were used to diagnose CKD.

In Silico Network Pharmacology and Molecular Docking Analysis

Identification of Drugs and Target Prediction

Furosemide, Febuxostat, and Torsemide are three drugs frequently employed in the treatment of CKD and were selected as the subject of a computational analysis. Their chemical and structural details were obtained from the PubChem database in SDF format.

Potential biological targets were predicted using Swiss Target Prediction (https://www.swisstargetprediction.ch/), which identifies likely macromolecular targets based on 2D/3D similarities with established ligands.

Screening of CKD-Associated Genes

Target genes associated with CKD were retrieved from the DisGeNET and GeneCards databases using the term "Chronic Kidney Disease." Overlapping targets between the drug-predicted genes and disease-associated genes were identified through Venn analysis (Venny 2.1.0).

Protein–Protein Interaction (PPI) Network

The shared targets were uploaded to the STRING database (https://string-db.org/) to create the PPI network with a confidence score of ≥0.9. The network was visualized and analyzed utilizing Cytoscape 3.10.3 software, and topological metrics such as node degree were used to identify hub genes via the CytoHubba plugin.

GO and KEGG Pathway Enrichment Analysis

The intersected targets were evaluated using ShinyGO to identify enriched Gene Ontology (GO) terms—biological process (BP), cellular component (CC), and molecular function (MF)—as well as KEGG pathways. Results were filtered using $p < 0.05$, and the top 10 items in each category were depicted.

Compound–Target–Pathway Network Construction

Interactions among drugs, targets, and pathways were integrated to develop a Compound–Target–Pathway (CTP) network using Cytoscape.

Molecular Docking

The top ten hub proteins (SRC, HSP90AA1, BCL2, PPARG, PTGS2, GSK3B, BCL2L1, MAPK14, RHOA, and MTOR) were selected for molecular docking using AutoDock Tools 1.5.7 and AutoDock Vina. The 3D protein structures were downloaded from the RCSB Protein Data Bank (PDB) **.**The polar hydrogen additions and the removal of water molecules were done to make proteins, and tuning of ligands was done to minimize the energy. Interaction analysis PyMOL interaction analysis with the docking results was used to evaluate the results in terms of binding affinity (kcal/mol).

RESULTS

Identification and Screening of Selected Drugs

All three medications including: Furosemide, Febuxostat and Torsemide were selected as the drugs that are commonly used to combine and treat CKD as well as related comorbidities. Table 1 reflects the pharmacological groupings, chemical specifications, and PubChem IDs.

These drugs were evaluated for their pharmacokinetic and pharmacodynamic relevance using ADMETLab 3.0, ensuring compliance with Lipinski's Rule of Five and acceptable safety profiles**.** The drugs and their characteristics are tabulated in Table_pow2. Furosemide (PubChem CID: 3440) is a sulphonamide diuretic, which is a loop diuretic. The most common manifestation is in the prevention of controlling blood pressure and to decrease the fluid or swelling overload of patients with CKD. Febuxostat (PubChem CID: 134018) is a thiazole derivative and an xanthine oxidase inhibitor that is used to treat inflammation caused by liver cirrhosis, heart failure and CKD. It also works well in controlling blood pressure. Torsemide (PubChem SID: 41781) is another sulo-nylurea analog of the loop diuretics.

Identification of Potential Therapeutic Targets

For the selected drugs, Swiss Target Prediction identified 18,571 potential drug targets with probability values greater than zero. CKD-associated genes were retrieved from the GeneCards and DisGeNET databases. Intersection of these datasets using Venn analysis revealed 205 overlapping genes, which represent the common targets potentially involved in CKD pathogenesis and drug action (Figure 1).

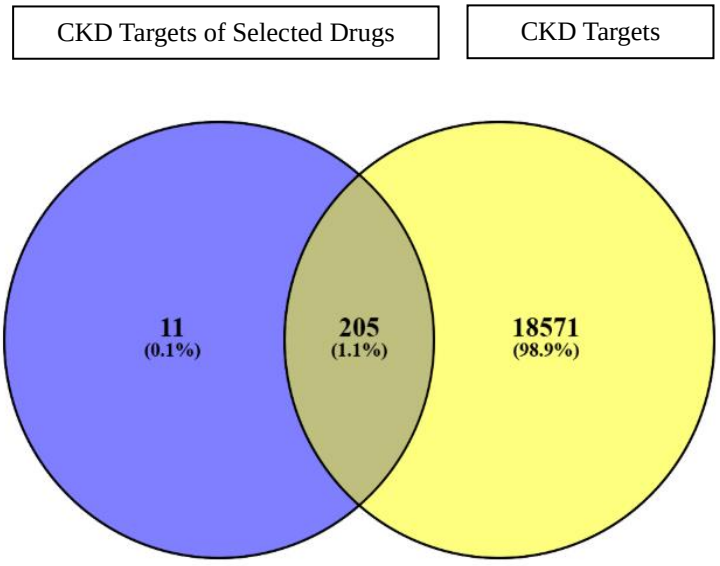

Fig. No. 1: Venn diagram of targets of drugs.

Protein–Protein Interaction (PPI) Network Construction

The 205 overlapping targets were imported into the STRING database for PPI network generation. The network contained multiple interconnected nodes, indicating strong functional relationships among targets. Topological analysis using the CytoHubba plugin in Cytoscape identified ten hub genes with the highest degree of connectivity, as shown in Table 2.

Table No 1: PDB ID's and standard drugs of hub protein targets of selected drugs against Chronic kidney disease.

S.NO	GENE	PROTEIN NAME	PDB ID
1	SRC	SRC Proto-Oncogene, Non-Receptor Tyrosine Kinase	6S4M
2	HSP90AA1	Peroxisome Proliferator-Activated Receptor Gamma	6KSQ
3	BCL2	BCL2 Apoptosis Regulator	6YBG
4	PPARG	Peroxisome Proliferator-Activated Receptor Gamma	6E5A
5	PTGS	Prostaglandin-Endoperoxide Synthase 2 (also known as COX-2)	5IKQ
6	GSK3B	Glycogen Synthase Kinase 3 Beta	6TCU
7	BCL2L1	BCL2 Like 1	6HJL
8	MAPK14	Mitogen-Activated Protein Kinase 14 (also known as p38α)	5O90
9	RHOA	Ras Homolog Family Member A	5JHH
10	MTOR	Mechanistic Target of Rapamycin Kinase	6M4U

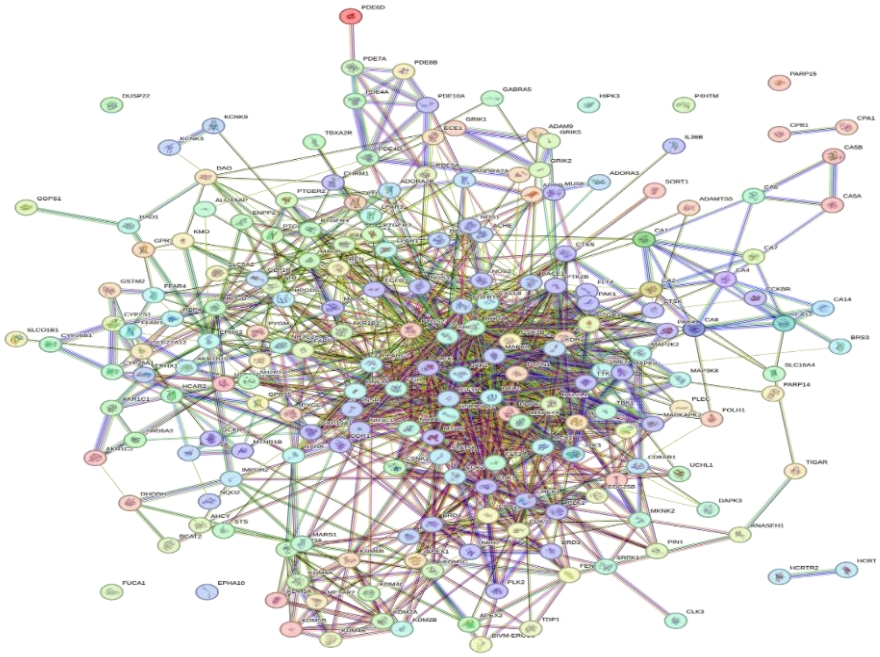

Fig. 2: STRING – PPI network for the 205 common targets between Chronic kidney disease compound targets and common drugs.

Using the cytohubba pug in top 10 proteins with highest degrees in the protein network were generated which could be major hubs responsible for the Chronic kidney disese. The Top 10 hub proteins are listed in table below

Table No 2: Top 10 hub gene targets from STRING PPI Network.

Rank	Gene	Score	Names of genes
1	SRC	71	SRC Proto-Oncogene, Non-Receptor Tyrosine Kinase
2	HSP90AA1	62	Heat Shock Protein 90 Alpha Family Class Member 1
3	BCL2	58	BCL2 Apoptosis Regulator
4	PPARG	54	Peroxisome Proliferator-Activated Receptor Gamma
5	PTGS2	52	Prostaglandin-Endoperoxide Synthase 2 (also known as COX-2)
6	GSK3B	45	Glycogen Synthase Kinase 3 Beta
7	BCL2L1	40	BCL2 Like 1
8	MAPK14	39	Mitogen-Activated Protein Kinase 14 (also known as p38α)
9	RHOA	37	Ras Homolog Family Member A
10	MTOR	37	Mechanistic Target of Rapamycin Kinase

GO and KEGG Pathway Enrichment Analysis

Gene Ontology (GO) and KEGG pathway analyses were performed using ShinyGO to explore the biological significance of the 205 overlapping genes.

The GO enrichment analysis showed that the most significant biological processes were response to chemical stimuli, protein phosphorylation, regulation of signaling, and cell communication. The major molecular activities were mainly linked with a kinase activity, oxidoreductase activity, nucleotide binding and catalytic activity on proteins. The large cellular structures that were observed were the nucleoplasm, synapse and plasma membrane region. Also, KEGG pathways analysis demonstrated a notable enrichment in metabolic pathways, PI3K-Akt signaling, PI3K-lipid/atherosclerosis pathways, cAMP signaling, Rap1 signaling, and EGFR resistance to tyrosine kinase inhibitors.

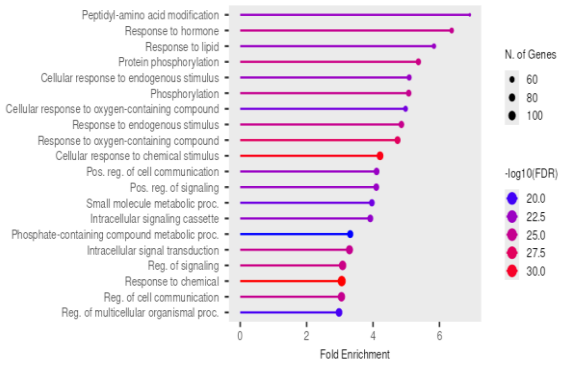

Fig No 3: KEGG Enrichment Analysis.

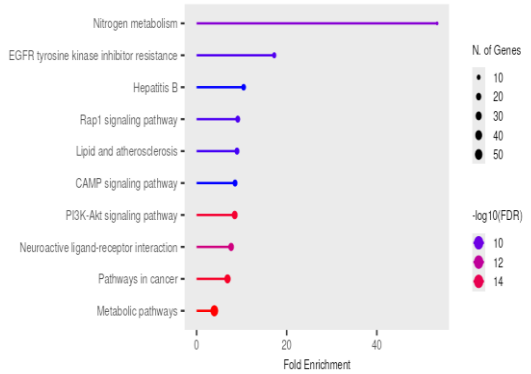

Fig No 4: Go Cellular components.

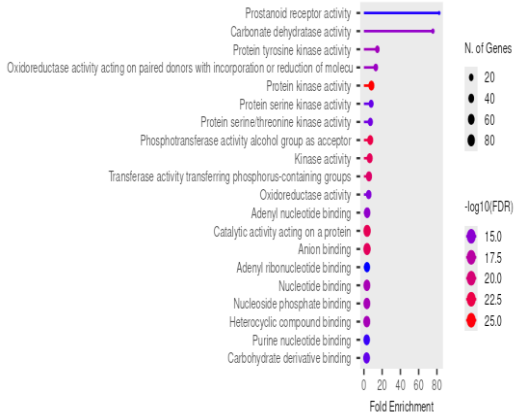

Fig No 5: GO Cellular Components.

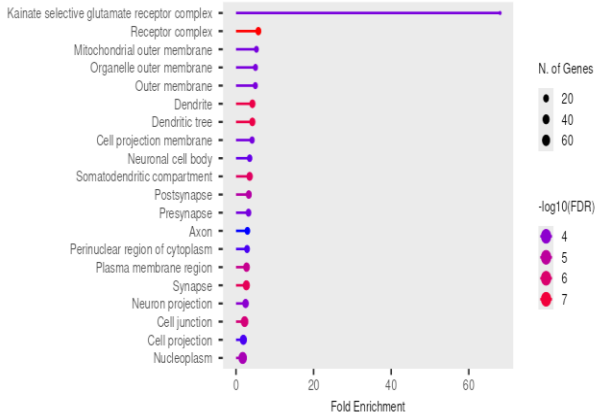

Fig No 6: GO Molecular Components.

Table no 3: Top 10 KEGG Enriched signaling pathways with related genes.

S.NO	PATHWAYS	GENES
1	Metabolic pathways	PDE10A CA5B CYP19A1 CYP26A1 DAO AKR1C1 AKR1C2 DHODH AHCY EPHX2 AKR1B1 FOLH1 CA14 HPGDS GSTM2 CYP2S1 HSD11B1 IMPDH2 STS MAOA MAOB MARS1 MIF NOS1 NOS2 NOS3 PDE4A PDE4B PDE4D PDE6D PDE7A ENPP2 HAO1 CYP26B1 AKR1B10 TIGAR PTGS2 PYGL PYGM BCAT2 SRD5A2 XDH CA1 CA2 CA4 CA5A CA6 CA7 CA9 CA12 KMO PDE8B PDE5A GGPS1
2	Pathways in cancer	CDK2 DAPK3 LPAR1 FGFR1 FGFR3 FLT3 FLT4 LPAR3 MTOR GSK3B GSTM2 HSP90AA1 IGF1R CXCL8 JAK2 JAK3 RHOA MET NOS2 PPARG MAPK8 MAPK9 MAP2K1 MAP2K2 PTGER2 PTGER3 PTGER4 PTGS2 BCL2 BCL2L1 RXRA STAT5B
3	PI3K-Akt signaling pathway	CDK2 CHRM1 LPAR1 FGFR1 FGFR3 FLT3 FLT4 LPAR3 MTOR GSK3B NR4A1 HSP90AA1 IGF1R INSR ITGB3 JAK2 JAK3 KDR MCL1 MET NOS3 PDPK1 MAP2K1 MAP2K2 BCL2 BCL2L1 RXRA
4	Neuroactive ligand-receptor interaction	CHRM1 ADORA1 ADORA2A ADORA2B ADORA3 LPAR1 LPAR3 GABRA5 GLP1R GPR35 GRIK1 GRIK2 GRIK5 NR3C1 HCRTR1 HCRTR2 MTNR1A MTNR1B PTGDR PTGER2 PTGER3 PTGER4 BRS3 TBXA2R CCKBR
5	CAMP signaling pathway	PDE10A CHRM1 ADORA1 ADORA2A GLP1R HCAR2 RHOA PAK1 PDE4A PDE4B PDE4D MAPK8 MAPK9 MAP2K1 MAP2K2 PTGER2 PTGER3
6	Lipid and atherosclerosis	MAPK14 TBK1 GSK3B HSP90AA1 CXCL8 JAK2 RHOA NOS3 PDPK1 PPARG MAPK8 MAPK9 BCL2 BCL2L1 RXRA SRC CASP1

7	Rap 1 signalling pathway	ADORA2A ADORA2B MAPK14 LPAR1 FGFR1 FGFR3 FLT4 LPAR3 IGF1R INSR ITGB3 KDR RHOA MET MAP2K1 MAP2K2 SRC
8	Hepatitis B	CDK2 MAPK14 PTK2B TBK1 CXCL8 JAK2 JAK3 MAPK8 MAPK9 MAP2K1 MAP2K2 BCL2 SRC STAT5B TYK2
9	EGFR tyrosine kinase inhibitor resistance	FGFR3 MTOR GSK3B IGF1R JAK2 KDR MET MAP2K1 MAP2K2 BCL2 BCL2L1 SRC
10	Nitrogen metabolism	CA14 CA1 CA2 CA4 CA5A CA6 CA7 CA9

Construction of Compound–Target–Pathway (CTP) Network

The selected drugs, predicted targets, and associated pathways were integrated to construct a Compound–Target–Pathway network using Cytoscape 3.10.3.

This comprehensive visualization demonstrated multi-target interactions of Furosemide, Febuxostat, and Torsemide with CKD-associated molecular pathways, confirming the polypharmacological nature of these agents.

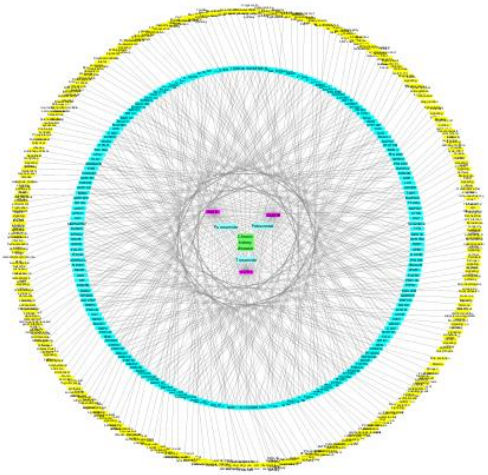

Fig. No 12: Cytoscape network of selected drugs with disease associated genes of CKD.

Interpretation of Network and Pathway Results

The PPI network constructed using the STRING database for 205 common targets showed dense interconnections, indicating strong functional relationships among CKD-associated proteins. CytoHubba analysis identified ten key hub genes (SRC, HSP90AA1, BCL2, PPARG, PTGS2, GSK3B, BCL2L1, MAPK14, RHOA, and MTOR), which are mainly involved in apoptosis regulation, oxidative stress, inflammation, and metabolic signaling.

KEGG pathway enrichment revealed significant involvement of metabolic pathways, PI3K–Akt signaling, pathways in cancer, neuroactive ligand–receptor interaction, cAMP signaling, and nitrogen metabolism, indicating that CKD pathogenesis involves dysregulated metabolic and signaling networks.

GO analysis showed that target genes were enriched in biological processes such as protein phosphorylation and cellular response to chemical and oxidative stimuli, localized primarily in the nucleoplasm, plasma membrane, and synaptic regions, and exhibited molecular functions including kinase, oxidoreductase, and nucleotide binding activities. Overall, the integration of network and pathway analyses suggests that the selected drugs may exert therapeutic effects in CKD by modulating key molecular pathways related to cell survival, inflammation, and metabolic regulation.

MOLECULAR DOCKING RESULTS

The 10 protein targets containing the most hits in the Cytoscape v3.10.0 program when searched with Cytohubba were subjected to molecular docking with three common pharmaceuticals with AutoDock Tools 1.5.7 and AutoDock vina. Ten CKD-associated target proteins (6S4M, 6KSQ, 6YBG, 6E5A, 5IKQ, 6TCU, 6HJL, 5JHH, 5O90, and 6M4U) were molecularly docked with furosemide, februxostat, and torsemide. The docking scores were between -4.318 and -8.805 kcal/mol that demonstrated a stable interaction of the ligands and the proteins. With the majority of targets, torsemide exhibited the highest binding affinities, followed by furosemide and febuxostat. Several hydrogen bond interactions and strong binding energies (≤ –5.0 kcal/mol) indicate that these medicines bind to CKD-related proteins effectively. All things considered, Torsemide showed the most positive and consistent interactions, suggesting its possible therapeutic value in the treatment of CKD.

Rank	Ligand (Drug)	Docking Score(kcal/mol)	Interpretation
1	Torsemide	–8.805	Highest binding affinity; strong and stable interaction with target protein
2	Torsemide	–8.311	Very strong binding; consistent high affinity across targets.
3	Febuxostat	–8.191	Strong and stable ligand–protein complex formation.
4	Torsemide	–7.733	Effective interaction with CKD-related protein.
5	Febuxostat	–7.733	Significant binding stability and favourable molecular interactions.

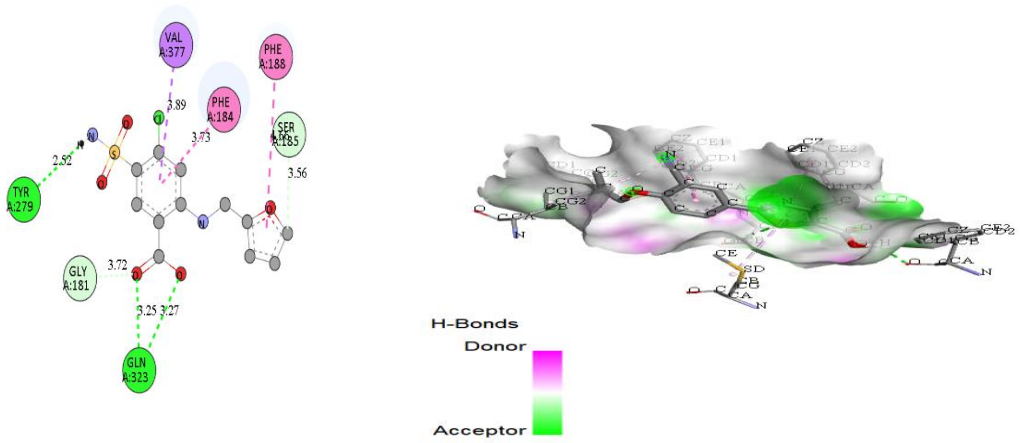

Fig no 2: 3D Docking pose and 2D interaction of Febuxostat against SRC (PDBID: 6S4M).

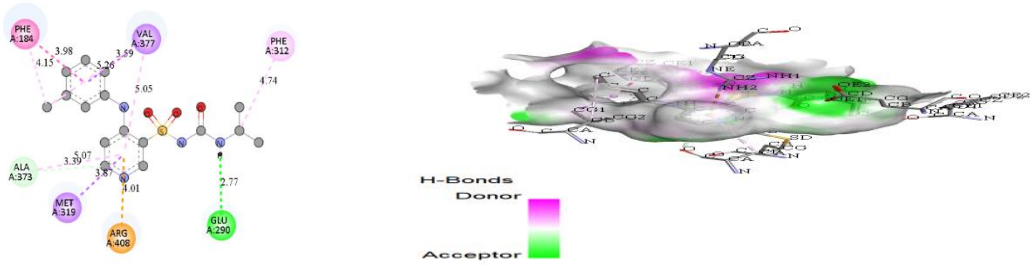

Fig no 3: 3D Docking pose and 2D interaction of Torsemide against SRC (PDBID: 6S4M).

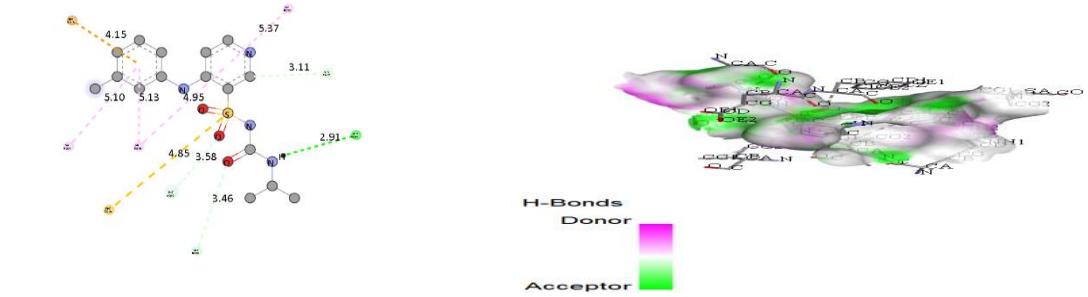

Fig no 4: 3D Docking pose and 2D interaction of Torsemide against BCL2 (PDBID: 6YBG).

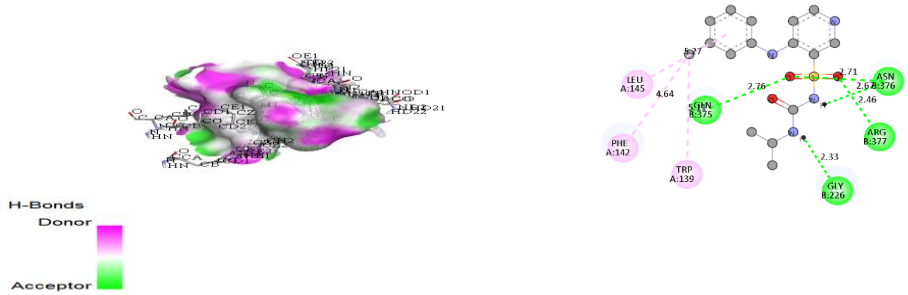

Fig no 5: 3D Docking pose and 2D interaction of Torsemide against PTGS2 (PDBID: 5IKQ).

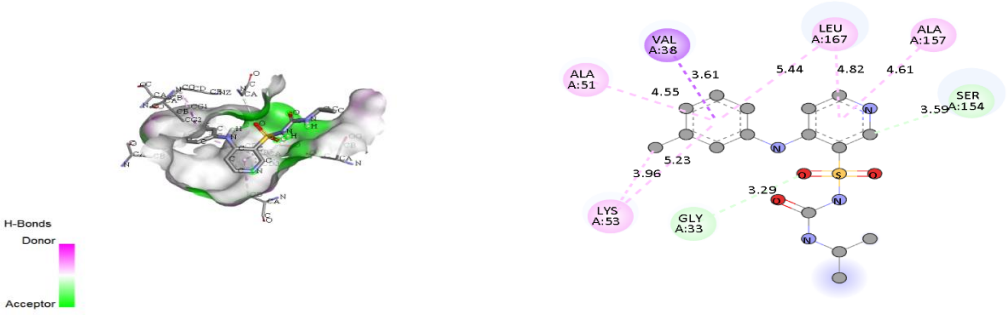

Fig no 6: 3D Docking pose and 2D interaction of Torsemide against MAPK14(PDBID: 5O900).

DISCUSSION

The present investigation utilized a combined network pharmacology and molecular docking strategy to elucidate the molecular mechanisms underlying the actions of three widely prescribed drugs in CKD management — Furosemide, Febuxostat, and Torsemide. An overlapping of CKD-related and drug-associated genes identifying 205 common targets was reported which highlights the multi target potential of these compounds. This is a systems-based solution because CKD pathogenesis is the product of intricate molecular networks that entail inflammation, oxidative stress, apoptosis, and fibrosis instead of the maladaptation of a single gene or receptor.[17]

Network Pharmacology Analysis

Ten major hub genes were identified in the protein-protein interaction (PPI) network created with help of STRING and Cytoscape SRC, HSP90AA1, BCL2, PPARG, PTGS2, GSK3B, BCL2L1, MAPK14, RHOA, and MTOR. These genes play vital roles in the regulation of inflammatory reactions, apoptotic signaling, and antioxidant response mechanisms. SRC was the non-receptor tyrosine kinase with the highest level of centrality. It has been linked to the stimulation of the activation of renal fibrosis and podocyte damage and the rejection of fibroblast activation and glomerular sclerosis in the event of its stimulation and inhibition, respectively.[18,20] The other important hub (HSP90AA1) is a molecular chaperone that stabilizes various signalling proteins such as Akt and HIF-1a which provide cytoprotective roles in the event of an oxidative or thermal stress.[4]

Moreover, BCL2 and BCL2L1 which are also apoptotic modulators in the mitochondrion were also amongst the top nodes-that reflect their functions of maintaining the integrity of the epithelial cells of the renal tubules and inhibition of apoptotic processes induced by oxidative stress. Since PPARG is an anti-inflammatory and antioxidant nuclear receptor, it is not novel that it is implicated in the process of metabolic and renal protection.[8]

Functional and Pathway Enrichment Analysis

Gene Ontology (GO) and KEGG analysis have been enriched, indicating a large amount of enrichment. The process involved the participation of signalling pathways:

- PI3K-Akt-mTOR signalling
- Rap1 signaling
- Nitrogen metabolism
- EGFR tyrosine kinase signaling.
- Metabolic and inflammatory processes.

PI3K-Akt-mTOR pathway is essential in controlling the growth of cells, energy generation, and the fibrosis of the renal cells. The hyperactivation of this pathway promotes glomerular hypertrophy and podocyte dysfunction, and inhibition by the pathway suppressed inflammation and fibrogenesis.[4,6] Rap1 signaling is another highly enriched pathway, and it is recognized to be in balancing the mitochondrial system and lowering the oxidative stress, which has been reported to alleviate the renal tubular injury.[19]

The increase in the pathways of nitrogen metabolism is related to uremic toxins accumulation and ammonia imbalance in the advanced CKD, whereas the stimulation of EGFR-related pathways reflects the role of growth factor signaling in kidney fibrosis and epithelial injur.[5]

Hub Gene Significance and Biological Implications

The list of recognized hub genes cover the SRC, MAPK14, MTOR and PPARG that are the center of an inflammation, apoptosis and metabolic regulation. SRC plays a fundamental role in phosphorylation and activation of the MAPK pathway by tyrosine and its inhibition has been demonstrated to prevent the deposition of the extracellular matrix and fibroblast stimulation, therefore, suppressing the renal fibrosis.[20] The pathway mediates the production of pro-inflammatory cytokines, including IL-6 and TNF-a, and glomerular damage, which is thwarted by the blockage of the pathway (Gui et al., 2020). MTOR is in charge of cell metabolism and autophagy; its hyperactivation causes proteinuria and hypertrophy, and its selective disactivation has renoprotective benefits.[6] PPARG is an anti-inflammatory

antioxidant anti-lipid and glucose metabolism transcriptional regulator. They found out that it was possible to prevent oxidative stress and the emergence of inflammatory cytokines in the kidney level using pioglitazone agonists.[13]

The combination of these findings indicates that the chosen drugs, in particular, Torsemide, might interact with key signaling proteins participating in the oxidative damage, fibrosis, and metabolic dysregulation, with a view to providing some understanding of the possible renoprotective action.

Molecular Docking Analysis

The molecular docking findings of the present study are in good agreement with the pharmacodynamics of the medicines. Febuxostat (-7.716 kcal/mol) and Torsemide (-8.805 kcal/mol) have strong binding affinities, which reflect long-lasting and physiologically significant interactions with the active pockets of BCL2, MAPK14, and SRC proteins. These findings are corroborated by clinical evidence that torsemide possesses anti-inflammatory and anti-fibrotic effects in addition to the effect of diuresis. Partly because of its increased tissue a recent randomized trial (TRANSFORM-HF) proved the superiority of the bioavailability torsemide medication with respect to clinical outcome and reduced hospitalizations as compared to furosemide.[14]

In a similar way, Febuxostat is well-known for its dual function as an antioxidant and xanthine oxidase inhibitor that reduces oxidative damage in renal cells.[16]

Despite being a diuretic, furosemide exhibited a moderate level of binding with HSP90AA1, suggesting a secondary function in stress response and protein homeostasis.[4]

These findings highlight the possibility that standard CKD drugs have pleiotropic protective effects, influencing inflammation and cell survival at the molecular level in addition to hemodynamic processes.

Summary

The network pharmacology study identified ten significant hub genes SRC, HSP90AA1, BCL2, PPARG, PTGS2, GSK3B, BCL2L1, MAPK14, RHOA and MTOR, which were found to have 205 overlapping targets between CKD related genes and pharmacological targets. There was an enrichment of major signaling pathways that are pertinent to the pathophysiology of CKD, including PI3K-Akt, Rap1, and nitrogen metabolism. Molecular docking revealed strong and consistent interactions with molecular docking especially with Torsemide which exhibited the highest binding affinities (-8.805 kcal/mol) with several target proteins. The implication of these interactions is that the drugs possess complicated therapeutic activities extending beyond their well-defined pharmacological activity, influencing the control of fibrosis, inflammation, and oxidative stress.

CONCLUSION

Among the studied medicines, Torsemide exhibited better molecular interactions with important CKD associated targets indicating more than diuresis. Drug use can be optimized, increased physician awareness can be increased, and improved reasonable, improve the outcome of the treatment, and reduce side effects. Network pharmacology and molecular docking evidence embrace the fact that conventional CKD drugs are multitargeting that might be utilized to expand the coverage of renal and slow the course of the disease.

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