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Review Article
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EMERGING THERAPEUTIC APPROACHES IN AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DIEASE

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Abstract
Mutations in PKD1 and PKD2 beget autosomal dominant polycystic order complaint (ADPKD), the most common renal inheritable complaint, leading to the dysregulation of renal tubules and the development of cystic growth that compromises order function. Despite significant advances in recent decades, there remains a considerable unmet clinical need, as current rectifiers are not effective at decelerating or halting complaint progression. Although preclinical beast models have been used considerably, the translatability of similar findings is uncertain and mortal-applicable complaint models are urgently demanded. Autosomal dominant polycystic order complaint (ADPKD) is the reported in 10 of end- stage order complaint (ESKD) cases and has an estimated frequence of 12.5 million cases worldwide across all races. There have been major advancements over the last two decades in understanding the pathogenesis and development of complaint- modifying treatment options for ADPKD, in nonsupervisory blessing of tolvaptan for ADPKD cases at threat of rapid-fire progression to order failure. In 2016, the position statement issued by the European Renal Association (period) was the first society- based recommendation on the use of tolvaptan and has served as a extensively used decision- making tool for nephrologists. Since also, considerable practical experience regarding the use of tolvaptan in ADPKD has accumulated.

Keywords: *ADPKD, polycystic order complaint, position statement, Autosomal dominant polycystic order, Biomarkers, habitual order conditions, prognostic, Tolvaptan, organoids; complaint modelling.*

INTRODUCTION

Autosomal dominant polycystic order complaint is a problem that affects the feathers. It's the inheritable reason for habitual order complaint. Autosomal dominant polycystic order complaint causes a lot of cases of end- stage complaint, up to 10 percent [1]. A study looked at eight studies and set up that Autosomal dominant polycystic order complaint affects 2.7 out of every 10,000 people. The main thing about Autosomal Dominant Polycystic order complaint is that it causes excrescencies to form in the feathers. People with Autosomal Dominant Polycystic order complaint frequently have problems like bleeding in their urine infections, in the excrescencies and the excrescencies can indeed burst. They can also get order monuments. Autosomal Dominant Polycystic order complaint is a problem worldwide. It's estimated that around 12.5 million people have it and it affects people of all backgrounds. This complaint is called a complaint. It starts with fluid- filled excrescencies in the feathers that just keep getting bigger over time. These excrescencies hurt the feathers. Make them not work duly by the time people are fifty or sixty times old [2]. There are about 0.6 million to 0.7 million cases of ADPKD in the United States. Two randomized clinical trials (RCTs) have shown a salutary effect of tolvaptan regarding the ADPKD- associated estimated glomerular filtration rate [eGFR] decline in cases with rapid-fire complaint progression. Considering the implicit downsides including its side goods and cost-associated with this treatment, the selection of cases who are most likely to show a positive benefit threat rate regarding this remedy-i.e. Individualities showing rapid-fire complaint progression are important and needed. Tolvaptan is a V2R antagonist that blocks vasopressin signal, a crucial motorist of tubercle growth in ADPKD due to the performing intracellular increase in cyclic adenosine mono phosphate. Importantly, only a subset of ADPKD cases suffers from rapid-fire complaint progression and will reach early order failure due to ADPKD, performing in the need for guidance regarding case selection. Following

the Working Group on Inherited order complaint (WGIKD) 2016 position statement [3]. In discrepancy, the original WGIKD position statement put the most weight on measured rapid-fire progression grounded on the literal decline in eGFR. This redounded in a more conservative algorithm that primarily recommended treatment for cases showing rapid-fire loss of order function in the once-the only real substantiation of factual rapid-fire progression. Still, since tubercle conformation precedes the decline in eGFR, cases despite a normal eGFR, and similar cases should n't be barred by a veritably restrictive algorithm. Likewise, vital information attained from the Duplication (Replicating substantiation of saved Renal Function A disquisition of Tolvaptan Safety and Efficacy in ADPKD) trials. autosomal Dominant Polycystic order complaint(ADPKD) is the most common inheritable order complaint, generally caused by pathogenic variants in the PKD1 and PKD2 genes. lately, fresh pathogenic variants in other genes have been linked, broadening the inheritable geography of ADPKD. Despite expansive exploration into remedial options, tolvaptan, a v2- receptor antagonist, remains the only United States (U.S.) food and medicine administration (FDA) approved complaint- modifying treatment for cases with a high threat of fleetly progressing order complaint. Also, multitudinous extrarenal man infestations are also common, including cardiovascular dysfunction. Intracranial aneurysms, and excrescencies in the liver and pancreas. Importantly, cardiovascular events are a leading cause of death in ADPKD cases, and hypertension(HT) may be a major contributing factor, as it presents in nearly 50 of cases before any loss in renal function, and in the late- stage cases, nearly all are affected [4]. The complaint is marked by the progressive development of order excrescencies, leading to order blow-up and eventual order failure (Kf). A DPKD accounts for 5-10 of all Kf cases, making it the fourth cause of Kf after diabetes mel litus, hypertension, and glomerulonephritis. Beyond sprat ney involvement, aDPKD is a systemic complaint with significant extrarenal instantiations, including liver and pancreatic excrescencies, valvular heart complaint, and an increased threat of intracranial Composition HISTORY entered 29 January 2025 revised 24 March 2025 accepted 7 april 2025 KEYWORDS aDPKD; polycystic order complaint; Polycystic liver complaint; tolvaptan; total order volume aneurysms. There are about 0.6 million to 0.7 million cases of ADPKD in the United States. Over the last decade, there have beenmajor advancements in understanding the pathogenesis and natural history of ADPKD, including identification of several complaint- causing mutations in genetically undetermined cases and development of complaint-modifying treatment op tions similar as tolvaptan. This review highlights the inheritable mutations associated with ADPKD, defines cases at threat of rapid-fire progression to ESKD, and focuses on the manage ment of ADPKD in the period of complaint- modifying agents. In the once 5 times the vasopressin V2 receptor (V2R) antagonist tolvaptan has become an important treatment option in the operation of cases with autosomal dominant polycystic kidney disease (ADPKD). This redounded in a more conservative algorithm that primarily recommended treatment for cases showing rapid-fire loss of order function in the once-the only real substantiation of factual rapid-fire progression trial in 2018 has allowed for an extension of eligibility criteria to aged cases and latterly- stage ADPKD.

EPIDEMIOLOGY

ADPKD affects up to 12 million people worldwide, with an estimated periodic prevalence of 2.5 cases per 100,000 individualities the frequence of aDPKD has been assessed using colorful styles, including population- grounded imaging studies, inheritable testing for pathogenic variants, and clinical individual criteria the complaint is observed across all races and races, although its frequence varies. advanced rates are reported in Black individualities (73 per 100,000) andnon-hispanic white individualities(63.2 per 100,000), compared to asian/ Pacific islanders(48.9 per 100,000) and Hispanics (39.9 per 100,000). Lanktree et al. examined two large whole genome sequencing (wGS) and whole exome sequencing (Wes) databases (gnomaD and Bravo) and estimated the likely true frequence of aDPKD to be roughly 1 in 1072 this figure is advanced than epidemiologic estimates (1 in 2000), pressing the implicit underestimation of milder or asymptomatic cases in large population studiesin the United States, aDPKD accounts for roughly 5 of new dialysis cases annually. Rates of Kf are specially advanced in males than ladies (8.2 vs 6.8 cases per million population, independently). Also, difference in dis ease progression are apparent;non-hispanic Black individualities with aDPKD reach Kf at a youngish age compared tonon-hispanic white individualities (54.4 vs 55.9 times, p< 0.0001). Contributing factors may include comorbid condi tions, similar as sickle cell particularity, and inheritable threat factors, including APOL1 threat alleles [5].

ETIOLOGY AND INHERITABLE

aDPKD is inherited in an autosomal dominant pattern, characterized by high penetrance but variable expression, with an equal coitus distribution complaint donation and inflexibility frequently vary indeed among family members, reflecting the interplay of inheritable, epigenetic, and environ internal factors. Studies reveal notable variability in aDPKD progression. identical halves differ by an normal of 2 times in order failure onset the maturity of a DPKD cases affect from pathogenic variants in two crucial genes PKD1 on chromo some 16 (account for 78 of cases) and PKD2 on chromosome 4 [15, 26] also, pathogenic variants in other genes, including IFT140, DNAJB11, GANAB, NEK8, and ALG5, ALG8, ALG9 contribute to a lower portion the PKD1 gene encodes polycystin- 1

(PC1), a multidomain membrane protein involved in extracellular relations and intracellular signaling pathways that regulate cell proliferation. PC1 is expressed in crucial structures intertwined in tubercle conformation, similar as order tubular epithelial cells, hepatic corrosiveness tubes, endothelial cells, and pancreatic tubes. PKD2 encodes polycystin- 2 (PC2), a calcium- regulated cation channel of the flash receptor eventuality(trP) family, set up in order tubular epithelial cells, vasculature, hepatic cells, brain, and placenta. Both PC1 and PC2 are localized tonon-motile primary cilia (supplemental figure S2), where their commerce is essential for PC1 stabilization, trafficking, and development [27], other genes intertwined in aDPKD affect PC1 trafficking or glycosylation in the endoplasmic reticulum crucial controllers of ciliary protein genes, impact aDPKD progression, with HNF1B being a recap factor that can upregulate the expression of multiple PKD- associated genes (e.g., PKHD1 and PKD2). Biallelic PKHD1 mutations beget autosomal sheepish polycystic order complaint (ARPKD) while monoallelic variants beget aDPKD with a milder donation. HNF1B variants, associated with autosomal dominant tubulointerstitial complaint (aDtKD), lead to HNF1B- related nephropathy. tSC genes are also important TSC1 and TSC2 mutations are intertwined in tuberous sclerosis complex(tSC), presenting with simple excrescencies and renal angiomyolipo mamas, or in TSC2/ PKD1 conterminous gene omission pattern, leading to early- onset cystic complaint. TSC mutations disrupts the mtor pathway, accelerati tubercle growth and aDPKD progression (Supplemental figure S3has been linked to worsened complaint inflexibility Inheritable Variant.

PKD1 inheritable variants are associated with earlier progression to end- stage order complaint compared with PKD2- 20 and abridging variants in PKD1 result in more rapid-fire progression than nontruncating variants [21, 28]. Cases with variants innon-PKD genes or no sensible variants generally have milder complaint and a better prognostic. PKD genotype alone does n't have sufficient prognostic delicacy to identify cases whose complaint will progress fleetly for remedy, but combining genotype with other labels similar as MIC can enhance prognostic models. Genetic testing is pivotal when a opinion is unclear, particularly in atypical cases, absence of family history, early or veritably early aDPKD (diagnosed before age 15 or perinatally over to 18 months), Kf without significant order enlarge ment, or when there's a distinction between eGfr decline and order tubercle burden testing is also essential in scripts similar as preimplantation inheritable opinion or assessing implicit youthful living order benefactors at threat of aDPKD. also, inheritable testing is recommended when extrarenal instantiations suggestive of runs other than aDPKD, linked in cases with atypical or unusual imaging findings and no family history [6].

CLINICAL INSTANTIATIONS, COMPLICATIONS AND OPERATION OF ADPKD

aDPKD presents with a wide diapason of renal and extrarenal instantiations, with utmost cases being asymptomatic until the third decade of life. Below is an overview of crucial instantiations, complications, and their managemen

A-Kidney Instantiations

Tubercle development, dropped eGFR, and order failure Cystogenesis begins in utero but generally remains asymptomatic until early majority order volume, particularly ht- tKv, is a strong predictor of complaint progression; [7]. variants affect in a milder course, with Kf being at 67.9 times, while PKD2 variants are associated with milder phenotype, developing Kf around 79.7 times. The minor and newer pathogenic variants associated with aDPKD have distinct phenotypes that lap with aDPKD- PKD1 and aDPKD-PKD2 for case, aDPKD- IFT140 is generally associated with increased tKv due to a many large excrescencies but has a low threat of order failure. aDPKD- DNAJ B11 involves only a many small excrescencies without tKv increase but carries a high threat of order failure latterly in life due to fibrosis interestingly, bilateral excrescencies involving three or further excrescencies are generally observed in collagenopathy, similar as those associated with variants in COL4A3 and COL4A4. these phenotypes may act features seen in rare aDPKD- associated genes like IFT140, DNAJB11, GANAB, ALG5, ALG8, and ALG9, suggesting lapping inheritable and phenotypic mechanisms. Liver function monitoring is essential due to the threat of reversible hepatoxicity the effect of tolvaptan is sustained and accretive and therefore it's recommended to start as beforehand as suitable in adult cases with aDPKD at threat of rapid-fire progression. the medicine is con traindicated in gestation, lactation, history of liver injury, hypovolemia, and urinary tract obstructions Supplemental table S2 details clinical trials exploring colorful remedial agents in aDPKD.> 60 mL/ min/ 1.73 m2 also, the duplication trial showed that tolvaptan braked the decline in eGfr by 35 over one time in cases with further advanced CKD(CrCl of 25 – 65 mL/ min/ 1.73 m2. an anal ysis by torres et al. on CKD G4 cases in the open marker trial(oLe) demonstrated that switching from placebo to tolvaptan significantly braked eGfr decline, with benefits extending to those with lower eGfr(15-24 mL/ min/ 1.73 m2) thus, tolvaptan remains effective indeed at eGfr situations as low as 15 mL/ min and is recommended until Krt is needed.

Hypertension

hypertension, defined as a blood pressure reading above 130/80 mmhg, affects up to 70 of aDPKD cases by age 30, frequently antedating significant order function decline Blood pressure (BP) control is a crucial element of operation, with targets acclimatized to patient age and order function. for utmost cases, maintaining BP 60 mL/ min/ 1.73 m2, a

stricter target of v a stricter target of< 110/75 mmhg is advised. The etiology of hypertension in aDPKD is multifactorial. Patient symptoms despite antibiotic remedy may bear Ct- guided aspiration for opinion and drainage.

Nephrolithiasis

Operation begins with icing acceptable hydration to adulterate urine, along with applicable pain control for obstructive or complex monuments, interventions similar as extracorporeal shock surge lithotripsy(eSwL), percutaneous nephrolithotomy, and flexible ureteroscopy are frequently needed still, the presence of large excrescencies in aDPKD complicate eSwL or percutaneous nephrostomy making flexible ureteroscopy a favored option due to its safety and expedited recovery. Uric acid monuments, although their use in this environment is n't widely accepted [8].

B-extrarenal manifestations

Liver cysts are the most common external manifestation of aDPKD, with prevalence increasing with age, reaching about 80–90% after age 35. risk factors for a higher liver cystic burden include estrogen-containing birth control, hormone replacement therapy, and greater number of pregnancies Polycystic liver disease (PLD) may impact quality of life, causing abdominal pain, bloating, early satiety, and dyspnea due to diaphragm compression. Complications such as cyst hemorrhage and infection can be detected by Ct or Mir Management is tailored to severity and includes pain relief, cyst aspiration with sclerotherapy, cyst fenestration, partial hepatectomy, or liver transplantation pancreatic cysts are less common (19% of patients) and are rarely symptomatic Seminal vesicle cysts occur in about 40% of male patients with aDPKD [9].

MANIFESTATIONS OF ADPKD IN CHILDREN

nonspecific symptoms in children include abdominal, flank, or back pain, cyst infections or bleeding Gross hematuria occurs in 10–14% of children before age 16, while symptoms like polyuria, urinary frequency, and enuresis (due to reduced urine concentrating ability) are seen in around 58% hypertension and mild proteinuria are com mon with 20–40% of children affected. hypertension accelerates kidney function decline and kidney growth Children with aDPKDveo face a higher risk for kidney function loss at a younger age. While most children maintain adequate kidney function into their 30s severe neonatal presentations can resemble arPKD NEK8 variants, particularly biallelic pathogenic variants, are linked to severe syndromic ciliopathies, whereas monoallelic variants in the kinase domain primarily affect the kidneys and resemble aDPKDveo.

MANAGEMENT OF ADPKD IN CHILDREN

Asymptomatic patients typically do not require treatment until adulthood, but early intervention is essential for those with hypertension or other symptoms tolvaptan use in children is under investigation. a recent phase 3 trial suggest it slows kidney volume growth and eGfr decline, though statistical significance was not achieved due to small sample sizes and short study durations. ambulatory blood pressure monitoring (aBPM) is preferred for diagnosing hypertension in children aged ≥5, as it detects isolated nocturnal hypertension raaS inhibitors are recommended to control blood pressure and slow kidney function decline Maintaining a healthy weight and reducing salt intake are additional strategies for optimal disease management Pravastatin was evaluated in a trial involving children and young adults (ages 8–22) with aDPKD. Over three years, it significantly reduced htTKV growth, although it had no impact on left ventricular mass index, urine micro albumin excretion, or kidney function.

DIAGNOSIS

ADPKD is primarily diagnosed through abdominal imaging, which typically reveals bilateral kidney cysts and kidney enlargement. Genetic testing is particularly valuable in specific scenarios, such as atypical presentations or when confirmation is required for family planning, risk stratification, or young potential kidney donors accurate diagnosis and follow-up are best managed by a multidisciplinary team that includes pediatric and adult nephrologists, geneticists, and radiologists experienced in aDPKD [Comprehensive counseling is essential to inform patients and families about the potential benefits and challenges associated with screening and genetic testing . Clinicians should test for aDPKD in individuals with a family history of CKD, Kf, PKD, or kidney cysts as well as in those with personal histories of CKD, hypertension, liver cysts, gross hematuria, or kidney stones. Magnetic resonance imaging (Mri) and computed tomography (Ct) scan are commonly used to confirm aDPKD diagnosis, measure height-adjusted total kidney volume (ht-tKv), as the presence of four or fewer cysts is sufficient to exclude the diagnosis in individuals with a family history of aDPKD non-contrast Mri is sufficient to provide most of the necessary diagnostic information. however, in cases where incidental masses or complex cysts are detected, contrast-enhanced Mri with gadolinium is recommended for further evaluation Liver cysts, which are present in over 85% of individuals with aDPKD by the age of 30, can support the suspicion for a cystic disease, particularly in patients without family history of aDPKD Both CCt and Mri are reliable for calculating tKv, differentiating cystic from non-cystic tissue, and assessing cyst burden. T2-weighted Mri is effective for visualizing kidney cysts, which appear hyperintense on t2 images, though its higher cost limits routine use non-contrast Ct is effective for detecting kidney stones but involves significant ionizing radiation, making frequent imaging impractical. Additionally, the use of iodinated contrast enhancement is typically avoided in moderate to advanced CKD Because of these risks; Mri is generally preferred over CCt for longitudinal monitoring, though Mri is unsuitable for detecting

kidney stones or nephrocalcinosis [10]. Opinion of ADPKD in children Discovery one or further order excrescencies in a child with a positive family history is largely suggestive of aDPKD. when results are equivocal follow- up studies are needed Mri offers advanced perceptivity but is frequently avoided in youngish children due to sedation conditions inheritable testing is recommended for atypical or early- onset donations and cases without family history of cystic feathers [11].

OPINION OF AUTOSOMAL DOMINANT POLYCYSTIC ORDER COMPLAINT AND SITUATIONS WHEN INHERITABLE TESTING IS NEEDED

Opinion in these situations, molecular inheritable testing would be prudent to confirm the opinion. As inheritable webbing is getting more readily available, the suggestions for testing will probably be more inclusive as these results enrich the prognostication in ADPKD [12].

TREATMENT

Tolvaptan, an oral vasopressin V2- receptor antagonist, is the only medicine approved for the treatment of ADPKD in grown-ups who are at threat of fleetly progressive order complaint. Its proposed medium of action is to decelerate tubercle growth by suppressing abnormally increased intracellular cyclic adenosine monophosphate situations in tubercle epithelial cells. Tolvaptan also reduced the prevalence of order pain and urinary tract infections. Although tolvaptan broke periodic TKV growth by 2.7, the effect on TKV tends to dwindle with time, making its effect on complications related to PKD mass effect uncertain. Analysis of the group aged than 55 times in Duplication did n't show a meaningful benefit of tolvaptan, but this was a small sample that had a slower drop in eGFR than the rest of the study population. nonetheless, in a pooled analysis of studies in cases with ADPKD who were aged than 55 times of age with CKD stage 3/4 and an eGFR drop ≥ 3 mL/ min/ 1.73 m2 per time, the periodic eGFR drop in cases taking tolvaptan was 1.66 mL/ min/ 1.73 m2 lower than with standard care over 3 times of follow- up [13]. therefore, there query as to the benefit of tolvaptan in cases aged than 55 times of age, and participated decision- making grounded on individual factors and cases' values is recommended. Likewise, cost remains a major hedge to the wide spread use of tolvaptan in ADPKD. Comforting about the benefits and threat of tolvaptan is essential. Adverse goods of tolvaptan include polyuria, pollakiuria, nocturia, increased thirst, and hepatotoxici but tend to ameliorate over time [14]. Disabled thirst response, cognitive vitiate ment, active hypernatremia, or urinary tract inhibition are relative contraindications to the use of vasopressin V2 receptor antagonists. In addition to consideration of complaint- modifying remedy, inhibition of the renin- angiotensin system, and blood pressure control, several life variations are recommended, although the substantiation base to support any of these is limited [15]. Operation of ADPKD Complications decelerating the progression of order excrescencies and the decline in order function, liver excrescencies develop in numerous cases, frequently with accompanying pain and gastrointestinal symptoms. Because the V2 vasopressin receptor is confined to the renal tubule, tolvaptan has no salutary effect on liver excrescencies [16]. Somatostatin and its analogues reduce intracellular cyclic adenosine monophosphate, including in the liver, and are effective at decelerating liver tubercle growth and reducing mass- related symptoms still, these analogues are frequently inadequately permitted as a result of adverse goods similar as hepatic tubercle infection, diarrhea, and hyperglycemia. PKD renal tubercle infections are resistant to treatment with antibiotic agents that are typically used for urinary in fections because the excrescencies do not communicate with the urinary tract. Positron emigration tomography reviews with f luorodeoxyglucose help distinguish tubercle infections from pyelonephritis. Lipid-answerable antibiotic agents like fluoroquinolones and trimethoprim-sulfamethoxazole are preferred for treating tubercle infections, with typical treat ment duration of 4 weeks [17].

BLOOD PRESSURE CONTROL

Hypertension is a common early incarnation of ADPKD due to renal ischemia from tubercle growth, cranking the renin- angiotensin system thus, angiotensin converting enzyme impediments or angiotensin receptor blockers are first- line curatives. There's no added benefit of combination remedy with angiotensin-converting enzyme impediments and angiotensin receptor blockers, cases treated to achieve this low blood pressure target had slower TKV growth than those treated to achieve a standard blood pressure target, although no benefit in terms of eGFR was sensible at 8 times. Blood pressure control also helps intracranial aneurysm growth and rupture, reduce left ventricular mass indicator, and drop albuminuria.

THE NEED FOR MOXIE REGARDING THE DECISION- TIMBER AND COMFORTING PROCESS

According to the vittles set by the EMA, tolvaptan treatment must be initiated and covered under the super vision of croakers with moxie in managing ADPKD and a full understanding of the pitfalls of tolvaptan remedy, including hepatotoxicity and monitoring conditions. The proposed algorithm provides guidance to help general nephrologists in the selection and operation of ADPKD cases on tolvaptan treatment. Still, it's judicious that small centers with only a many ADPKD cases that are potentially eligible for tolvaptan treatment take the occasion to consult an educated center regarding the selection of individual cases; including evaluation of MRIs, patient comforting and the management of side effects this algorithmaims

to provide substantiation- grounded medical guidance agreement [18].

CONCLUSION

In the era of disease-modifying treatments intended to slow the disease progression of ADPKD, five main points need to be addressed in an individualized fashion: 1) con firm the diagnosis of ADPKD by ensuring the cystic burden matches the observed kidney function; 2) assess the risk of rapid progression using available biomarkers such as age and htTKV; 3) implement renal protective measures for all ADPKD patients; 4) evaluate eligibility for disease-modifying treatments such as tolvaptan by discussing the risks, benefits, and patient preference; and 5) implement safe prescription of tolvaptan based on regulatory guidance for serial liver function testing

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REFERENCES

1. Torres VE, Harris PC, Pirson Y. Autosomal dominant polycystic kidney disease. Lancet. 2007;369(9569):1287-1301. doi:10.1016/S0140-6736(07)60601-1.
2. Solazzo A, Testa F, Giovanella S, et al. The prevalence of autosomal dominant polycystic kidney disease (ADPKD): a meta-analysis of European literature suggests underdiagnosis. PLoS One. 2018;13(1):e0190430. doi:10.1371/journal.pone.0190430.
3. Mufti UB, Nalagatla SK. Nephrolithiasis in autosomal dominant polycystic kidney disease. J Endourol. 2010;24(10):1557-1561.
4. Chapman AB, Devuyst O, Eckardt KU, et al. Autosomal-dominant polycystic kidney disease (ADPKD): KDIGO executive summary. Kidney Int. 2015;88(1):17-27. doi:10.1038/ki.2015.59.
5. Prasanthi G, Chandu BR, Pradeep Kumar Y, Swarnalatha D, Gopinath. Chemical pharmacology of khat leaves. J Glob Trends Pharm Sci. 2014;5(4):2024-2029.
6. Torres VE, Harris PC. Strategies targeting cAMP signaling in the treatment of polycystic kidney disease. J Am Soc Nephrol. 2014;25(1):18-32. doi:10.1681/ASN.2013010039.
7. Hwisa NT, Katakam P, Chandu BR, Abadi EG, Shefha EM. Comparative in vivo evaluation of three types of honey on topical wound healing activity in rabbits. J Appl Pharm Sci. 2013;3(8):139-143.
8. Torres VE, Chapman AB, Devuyst O, et al. Tolvaptan in later-stage autosomal dominant polycystic kidney disease. N Engl J Med. 2017;377(20):1930-1942. doi:10.1056/NEJMoa1710030.
9. Nama S, Chandu BR, Awen BZ, Khagga M. Development and validation of a new RP-HPLC method for the determination of aprepitant in bulk and pharmaceutical dosage forms. Trop J Pharm Res. 2011.
10. Gade R, Aynampudi A, Makineni A, Murthy TEGK, Rao CB, Nama S. Design and development of pravastatin sodium fast dissolving films from natural mucilage of Ocimum basilicum seeds. Int J Pharm Res Rev.
11. Chebib FT, Torres VE. Assessing risk of rapid progression in autosomal dominant polycystic kidney disease. Am J Kidney Dis. 2021;78(2):282-292. doi:10.1053/j.ajkd.2020.12.020.
12. Ecder T, Schrier RW. Cardiovascular abnormalities in autosomal-dominant polycystic kidney disease. Nat Rev Nephrol. 2009;5(4):221-228. doi:10.1038/nrneph.2009.13.
13. Ravikumar K, Chandu BR, Challa BR, Chandrasekhar KB. Method development and validation of almotriptan in human plasma by HPLC-MS/MS. Sci Pharm. 2012;80(2):367.
14. Fick GM, Johnson AM, Hammond WS, Gabow PA. Causes of death in autosomal dominant polycystic kidney disease. J Am Soc Nephrol. 1995;5(12):2048-2056.
15. Kelleher CL, McFann KK, Johnson AM, Schrier RW. Characteristics of hypertension in young adults with autosomal dominant polycystic kidney disease. Am J Hypertens. 2004;17(11):1029-1034.
16. Dey B, Hwisa NT, Khalf AMM, Mitra A, Katakam P, Chandu BR. Pharmaco-epidemiological studies on self medication and drug utilization pattern in chronic diseases via prescription auditing. Int J Sci Res Knowl. 2013;1(11):464.
17. Bethapudi V, Pasam NA, Velchuri S, Chandu BR. Dendrimers-emerging polymers for drug delivery and its future prospects. Res J Pharm Biol Chem Sci. 2012.
18. Rani CHU, Sumalatha G, Rao CHB, Varalakshmi TN. Alzheimer's disease-pharmacotherapeutic interventions. Int J Pharm Chem Sci. 2013.