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Original article

Diet and polycystic kidney disease: A pilot intervention study

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SUMMARY

Background & aims: Dietary sodium, protein, acid precursors, and water have been linked to cyst growth in polycystic kidney disease; yet, no studies in patients have examined the feasibility of using a dietary intervention that controls all of these factors. The aim of this study was to determine if a diet, appropriate for persons of most ages, reduces the excretion of sodium, urea, acid, and decreases mean urine osmolality while gaining acceptance by patients with autosomal dominant polycystic kidney disease (ADPKD).

Methods: Twelve adults with ADPKD enrolled in a pre-post pilot feasibility study and served as their own controls. Individuals consumed their usual diet for one week then for four weeks followed an isocaloric diet lower in sodium and protein and higher in fruits, vegetables, and water. Three-day diet records and two 24-h urine samples were collected at baseline, week 2, and week 4 visits; blood pressure, weight, and serum were obtained at all three visits. A modified nutrition hassles questionnaire was completed on the last visit.

Results: During the dietary intervention, subjects (n = 11) consumed less sodium, protein, and dietary acid precursors 36%, 28%, and 99%, respectively, and increased fluid intake by 42%. Urinary sodium, urea, net acid excretion, osmoles, and osmolality decreased 20%, 28%, 20%, 37%, and 15%, respectively; volume increased 35%. Urine changes were in accord with the diet record. Ninety-one percent of participants reported that none of the hassles were worse than "somewhat severe", and most participants felt "somewhat confident" or "very confident" that they could manage the new diet.

Conclusions: A majority of adult patients with ADPKD successfully prepared and followed a composite diet prescription with decreased sodium, protein, acid precursors, and increased fluid intake. This trail was registered at ClinicalTrials.gov (NCT01810614).

Abbreviations: ADPKD, autosomal dominant polycystic kidney disease; AI, adequate intake; AMDR, acceptable macronutrient dietary range; AVP, arginine vasopressin; BMI, body mass index; BUN, blood urea nitrogen; CRISP, consortium for radiologic imaging of polycystic kidney disease; DRI, dietary recommended intake; eGFR, estimated glomerular filtration rate; GFR, glomerular filtration rate; NAE, net acid excretion; NE, Not established; NEAP, net endogenous acid production; NDSR, Nutrition Data for System Research; NHQ, Nutrition Hassles Questionnaire; OA, organic acids; PKD, polycystic kidney disease.

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1. Introduction

Autosomal dominant polycystic kidney disease (ADPKD) is a genetic disorder that affects ~600,000 individuals in the United States and ~12.5 million people worldwide [1]. The clinical hallmark of ADPKD is the development of cysts in individual renal tubules that enlarge progressively throughout life. Although cysts begin forming during fetal development, the glomerular filtration rate (GFR) usually stays within an apparently normal range for several decades owing to compensatory hyperfiltration [2]. Meanwhile, cysts increase in number and volume, causing serious injury to blood vessels and renal tubules. Recent evidence indicates that kidney volume predicts the likelihood of developing renal insufficiency over time, suggesting that the growth of cysts is linked to declining kidney function [3].

The disease phenotype is highly variable causing differing rates of cyst growth even among members of the same family [4]. Dietary factors appear to account for some of the variability in this rate of renal enlargement; a process that appears to be hastened by the excessive intake of salt and animal-sourced protein [5,6]. Additionally, increased urinary acid excretion accelerates cyst growth in experimental animals and leads to a more rapid decline in kidney function in patients with moderately advanced chronic progressive renal diseases, including ADPKD [5,7–14]. Acid excretion is largely driven by high intakes of animal-sourced dietary protein, but can be reduced by base-producing fruits and vegetables [15]. Increasing fluid intake has also been shown to reduce kidney weight (% of total body weight) by 27–30% by lowering plasma levels of arginine vasopressin (AVP); a result achieved when reaching a urine osmolality of <290 mosm/kg H_2O [16]. In view of this evidence, we aimed to determine if a relatively complex dietary prescription targeting dietary salt, protein, acid precursors, and fluid intake would be meaningfully adopted by adult patients with ADPKD.

2. Materials and methods

2.1. Study population

Twelve subjects with a certain diagnosis of ADPKD confirmed by family history, magnetic resonance, computed tomography or ultrasound imaging were enrolled [1]. Subjects were recruited from the University of Kansas Medical Center Polycystic Kidney Disease clinic during routine visits between May 2013 and January 2014. Inclusion in the study required a blood pressure <135/85 mmHg, stable weight and clinical biochemistry, a diet history of >30 mEq/day of net endogenous acid production (NEAP), and an estimated GFR (eGFR) of >30 ml/min/1.73 m^2 based on the MDRD-EPI equation [17]. Subjects were excluded if they used pharmaceuticals or dietary restrictions/enhancements for preexisting medical conditions not associated with standard of care for ADPKD or if they had been prescribed medications that affect acid/base status. A physical examination was performed by a physician to confirm medical appropriateness to participate. Written informed consent was obtained prior to study enrollment. This study was approved by the Human Subject Committee at the University of Kansas Medical Center. This trial was registered at clinicaltrials.gov (NCT01810614). A physician co-investigator monitored data and safety.

2.2. Study design

Subjects were enrolled for a five-week study that included an enrollment visit, three study visits (baseline, 2 weeks, 4 weeks) and two follow-up phone calls (Fig. 1). Study visits occurred in the clinical and translational science unit. Each subject submitted a three-day diet record (three days prior to study visits regardless of weekday or weekend), two consecutive 24-h urine collections (two days prior to study visit), and a study nurse recorded blood pressures, anthropometrics, and drew blood samples at each visit. All subjects received instructions on how to modify their diet and served as their own controls in this pre-post pilot feasibility study.

2.3. Three-day diet records

Diet records included a detailed accounting of all food and beverage consumed, the amounts consumed, the method of food preparation, and, if homemade, the recipe that was used. The amount of food consumed was measured by weight on an electronic food scale or by typical volumetric household measures. If neither of these methods of measurement were available, participants were asked to estimate portion sizes using picture books that were provided. Diet records were analyzed at each visit using the Nutrition Data for System Research (NDSR version 2012) [18]. To improve collection of dietary data, probing techniques and a multiple pass system were used during study visits to gather information that subjects may have omitted or recorded incorrectly [19].

2.4. Urine measurements

Participants were instructed at the enrollment visit on how to collect 24-h urine samples. Instructions included detailed methods for properly recording start and stop times and when to switch to the second 24-h collection. Written instructions were also provided. Timed 24-h urine collections were completed on the two days prior to each visit and coincided with the last two days of the diet record. Urine collections were kept on ice or placed in a refrigerator throughout the duration of each timed collection. Urine volume was determined from the weight of the collected sample. Aliquots of urine were centrifuged, and stored immediately at −20 C for the later measurement of urinary acid excretion [20]. The remaining urine was sent immediately to the University of Kansas Hospital lab for analysis of electrolytes, urea, creatinine, bicarbonate, and osmolality.

2.5. Other measurements

Height was measured without shoes using a stadiometer and weight was recorded with participants without shoes in street clothes. Subjects were supine for the measurement of blood pressure. Electrolytes, blood urea nitrogen (BUN), creatinine, uric acid, glucose, and bicarbonate were determined in serum samples by the hospital laboratory.

2.6. Diet and urine calculations

2.6.1. Diet

Three-day diet records were used to calculate net endogenous acid production from the formula NEAP = PRAL + organic acids (OA) and the mean recorded. NEAP is used to predict the amount of acid excreted in the urine [21,22]. The first term, PRAL, estimates the potential net acid or base contribution a food or liquid would make when combusted by whole body metabolism [23]. The daily PRAL was calculated from all food and drink consumed over the day by estimating the dietary intake of anions (phosphorus and sulfur) and cations (potassium, calcium, and magnesium) from diet records using nutrient profiles from NDSR; sulfur was estimated based on the normative content of methionine and cysteine derived from protein in the diet. The fractional absorption of nutrients by the intestines, the dissociation of phosphate, and ionic valences are all taken into account in Remer's method for determining PRAL [21,23]. Daily consumption of these anions and cations was entered

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