

Dietary Therapy for Children with Congenital Solitary Functioning Kidney

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Abstract

Contextual: Renal injury, proteinuria and hypertension, are significances of inherited private operative kidney (CSFK). Renal injury appears around 10 years of age and required dialysis by the age of 30 years. Small protein consumption lessened renal injury in animal untried models, uninephrectomized patients and kidney transplant donors and recipients. Low salt intake facilitates blood pressure control.

Objective: Relevant data concerning CSFK patients had not been described thus far. We decided to look at long preventing effect of protein restriction and low-sodium diet on proteinuria, kidney function and hypertension, during this distinct population. Methods: Twenty eight children with CSFK were included during a prospective observational exposure series. Succeeding parents' arrangement, protein and salt limit: 0.85x recommended daily allowance (RDA) was started under dietitian supervision. Acquiescence confirmation was achieved by: Monitoring urinary urea nitrogen and body weight; and measuring 24 hour urine sodium. Complement (8-22 years and despicable 15.8 years) comprised clinical, growth and developmental assessment, renal function and urine protein excretion.

Patients and Methods:

Twenty-eight children were included in our study. All of them had been mentioned our pediatric nephrology outpatient clinic over a period of 14 years for observation due to ultrasonography findings of CSFK. Doubtful diagnoses were confirmed by renal dynamic scan. Protein and salt restriction: 0.85 × recommended daily allowance (RDA) was started under a dietitian's continuous supervision in every patient since weaning (breastfeeding or industrial formulae). Compliance verification of (a) protein and (b) salt restrictions was performed by (a) monitoring urinary urea nitrogen (g/24 h) and weight, consistent with the subsequent equation:

$$\text{Daily protein intake(g)} = [(\text{Urinary urea nitrogen(g/24h)} + 0.031 \times \text{Body weight(kg)}) \times 6.25]$$
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The follow-up included twice a year visits within the first 3 years then once-a-year visit. Twenty-four-hour urine collections were done from the age of three years. Just in case of doubtful accuracy of the gathering, another one was performed, after further explanation. Patients and fogeys were evaluated and instructed on each visit by pediatric nephrologists and dieticians. The entire period of follow-up was 22 years. Twenty-three completed the study, as young adults.

Statistical Analysis:

Analysis of knowledge was administered using SPSS 11.0 statistical analysis software (SPSS Inc, Chicago, IL). Outcome variables (serum creatinine, rates of RDA for protein intake and for salt intake) as descriptive statistics were calculated and reported as mean ± variance.

Results:

Baseline data showed normal physical assessment, blood pressure kidney functions and urinalysis. There was one patient with ipsilateral kidney malformation and two with systemic pathologies. Adherence to dietary restrictions: 89 ±8% for protein and 93 ±5% salt. At the top of follow up: two patients had chronic renal disorder (CKD) grade I. Not one person had hypertension, proteinuria or growing and enlargement worsening.

One child was uninephrectomized (6 months old), due to UPJ stenosis, which caused severe hydronephrosis and hydroureter (right kidney). This kidney, actually, wasn't working at birth.

Adherence to the diet regime is expressed because the rate of protein and salt restriction targets, consistent with urine

measurements described within the Methods section. Visit numbers represent 4- to 5-year intervals between each, when 1 is that the first presentation with the diagnosis of CSFK. Adherence to dietary restrictions was $89 \pm 8\%$ for protein and $93 \pm 5\%$ for salt.

The number of CSFK patients in our one-center observation study was larger than expected. as long as the annual birth rate in our municipal area was 20/1000 at the start of the study (30% to 80% quite within the Western world and Asia) leads us to suppose that this condition is more frequent than had been considered thus far (around 1/180 in our regional population vs 1/2031). it's according to our assumption that "real" incidence within the past was also much greater (see the Introduction section).

Arguably, lack of an impact group may be a certain limitation of our study. However, studies summarizing unfavorable outcome of youngsters with CSFK do exist, during which none deals with preventive or preservative therapeutic strategy. They can be considered historical controls for our work, considering identical population characteristics and nutritional habits, supported Israeli data of daily consumption of protein and salt among children. Furthermore, identified as exposure series observation (exposure = CSFK), its favorable final outcome is valid: only 8.6% with mild renal injury, none had proteinuria, hypertension, and growth-retardation. One cannot ignore the effect of the intervention.

The 2 exceptional patients who developed chronic renal disorder (CKD), grade I, represents the known risk factor for renal injury, CAKUT, in patient number 19 and lack of enough observation and nutritional guidance thanks to a comparatively short time of follow-up, 8 years, since the age of 17 years (patient number 7). Their urine protein excretion (<400 mg/24 h) was not influenced by exercise (15 mg/dL, both in resting condition and after exercise), as evaluated separately, in parallel to the aforementioned results.

We did not find a correlation between imaging results (including CRG or normal sized kidney) and kidney injury at the end of follow-up in our patient. All of them showed normal development, weight, and height gain.

Taken together, we conclude that strict follow-up with controlled dietary supervision for mild protein and salt restriction prevented kidney injury proteinuria and hypertension in CSFK. This therapy had no detrimental influence on growth and development.

Conclusion: Severe follow-up and measured dietary management for slight protein and salt constraint avert kidney injury proteinuria and hypertension in CSFK, without insults on growth and development.

Mechanisms contribute to the overall cancer preventive effects of dietary phytochemicals. Signal transduction pathways are potential molecular targets for chemoprevention by dietary phytochemicals. Increasing expression of detoxifying enzymes and/or antioxidant enzymes inhibit the cell cycle progression and cell proliferation, induce the differentiation and apoptosis, inhibit the expression and functional activation of oncogenes, increase the expression of tumour-suppressor genes, and inhibit angiogenesis and metastasis by modulating cellular signalling pathways. Dietary supplements and/or food fortification supported food by-product could also be alternative for above-mentioned healthy constituents.