Quality or Quantity of Proteins in the Diet for CKD Patients: Does “Junk Food” Make a Difference? Lessons from a High-Risk Pregnancy

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Key Words

Chronic kidney disease · Diet · Pregnancy

Abstract

Background: How to manage patients with severe kidney disease in pregnancy is still a matter of discussion, and deciding if and when to start dialysis is based on the specialist's experience and dialysis availability. The effect of toxic substances usually cleared by the kidney may be more severe and readily evident. The review, and related case, underlines the importance of considering the presence of additives in food in delicate conditions, such as CKD pregnancy. **The Case:** A 39-year-old indigenous woman from a low-resourced area in Mexico was referred to the obstetric nephrology at 25 gestational weeks because of serum creatinine at 3.6 mg/dL, hypertension on low-dose alpha-methyl-dopa, and nephrotic-range proteinuria. Kidney ultrasounds showed small poorly differentiated kidneys; foetal ultrasounds detected a female foetus, normal for gestational age. The patient's baseline protein intake, which was esti-

mated at 1.2–1.3 g/kg/day, was mostly of animal-origin (>70%) poor-quality food (“junk food”). In the proposed diet, protein intake was only slightly reduced (1.0–1.2 g/kg/day), but the source of proteins was changed (only 30% of animal origin) with attention to food quality. A remarkable decrease in BUN was observed, in concomitance with adequate dietary follow-up, with rapid rise of BUN when the patient switched temporarily back to previous habits. A healthy female baby weighing 2,460 g (11th centile for gestational age) was delivered at 37 gestational weeks. **Discussion and Literature Review:** While data on patients with chronic kidney disease are scant, the long list of contaminants present in food, especially if of low quality, should lead us to reflect on their potential negative effect on kidney function and make us realize that eating healthy, unprocessed “organic” food should be encouraged, in delicate conditions such as pregnancy and breastfeeding and for young children, in particular when kidney function is failing. The case herein described gave us the opportunity to reflect on the importance of diet quality and on the potential risks linked to food additives, many of which, including phos-

phates and potassium, are not declared on food labels, while others, including dyes, antioxidants, thickeners, emulsifiers, and preservatives, are qualitatively, but not quantitatively, reported.

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Background

The early phases of human development, and the diseases occurring in these phases, are increasingly being recognized as sources of information about the future health of the mother and child [1–4]. In individuals without a known kidney disease, the hypertensive disorders of pregnancy point to an increased risk of future cardiovascular diseases and end-stage kidney disease [5, 6]. The hypertensive disorders of pregnancy may also often be the first indication of pre-existing CKD [7]. Conversely, patients with known CKD have an increased risk of hypertensive disorders in pregnancy due to not fully understood interactions between placental development, renal hyperfiltration, and reduction in nephron number [8]. How to manage patients with severe kidney disease in pregnancy is still a matter of discussion, and deciding if and when to start dialysis is based on the kidney specialist's experience and dialysis availability [9, 10]. While in highly resourced settings indications tend to favour an early start of dialysis, in poorly resourced ones, modulation of dietary habits may represent a safe alternative at least for a part of the patients [11–13]. Interestingly, in previous experiences, a carefully monitored moderate protein restriction was associated with preserved intrauterine growth [14]. Data from the overall population support these findings, suggesting that plant-based diets are safe in pregnancy and that adding proteins or amino acids to already protein-rich diets is almost paradoxically associated with foetal growth impairment [15–17].

Pregnancy in CKD is often psychologically stressful for the mother, the family, and caregivers. From the clinical point of view, pregnancy-induced hyperfiltration may increase proteinuria even in the absence of placental dysfunction [18, 19]. Likewise, the effect of toxic substances may be more severe, and more easily detected, due to its effects on maternal kidney function and on foetal health; this is the case of some environmental pollutants, whose association with pregnancy outcomes can be taken as a proof of their effect on health [20, 21]. The importance of considering the presence of additives in food in delicate conditions, such as CKD pregnancy, and the

broader lessons we can learn from this experience are the issues we wish to discuss in this report.

In the nutritional management of advanced CKD, our attention progressively shifted from a quantitative approach, based on macronutrient intake (proteins, carbohydrates, and lipids), to a qualitative one, considering type of nutrient (plant-based vs. animal-based proteins, saturated or unsaturated fatty acids, short- vs. long-chain carbohydrates, etc.) as well as how nutrients are combined (Mediterranean diet, cafeteria diet, etc.) [22–25]. Furthermore, the problem of contaminants, preservation agents, trace elements, and pesticides is rapidly emerging, and their toxic effect is increasingly being recognized [26–28]. In this regard, CKD patients are a particularly interesting population, both because kidney disease may reduce excretion and enhance the toxicity of added substances and because a negative effect on the kidney function may be more readily evident in the presence of a reduced glomerular filtration rate, once the “buffering effect” of the renal functional reserve is offset.

The combination of advanced CKD and pregnancy is, therefore, not only a nutritional challenge but also a valuable occasion for highlighting, as though under a magnifying lens, the effect of dietary changes on health. In the case described, a pregnant woman with advanced CKD, was switched from “junk food” to a healthy diet and was able to avoid dialysis during pregnancy.

The Case

A 39-year-old indigenous woman affected by advanced CKD, born in Zacatepec, Oaxaca, and living in a remote low-resourced area in Mexico, was referred to the obstetric-nephrology department of the National Institute of Perinatology (INPER) in Mexico City in September 2019, at 25 gestational weeks. The woman, who lived with her husband's parents, was illiterate, as are many women of her age in remote rural areas, and had never worked outside her home. The pregnancy was her second one, but there were no available written records regarding her first pregnancy, which occurred in 2014. She recalled that she had delivered at term (41 weeks) by caesarean section because of preeclampsia. The baby, a girl, was born healthy and had regularly attained all the normal development milestones. The patient did not remember her birth weight, but she was apparently normal for gestational age.

While she was not aware she had CKD, upon careful inquiry, it seemed likely that kidney disease had been de-

tected during the previous pregnancy, since, after delivery, she had been told that she should have periodic blood tests to determine whether there were problems in kidney function, which, for economic and logistic reasons, she had not performed. She was also advised to curtail her consumption of meat and had been prescribed erythropoietin.

She reported normal blood pressure, but controls were not frequent. She had also been advised that a future pregnancy would be a risk for her health.

When she became pregnant again, 5 years later, in March 2019, she moved to her mother-in-law's home because it was nearer to the nearest hospital (2 h away instead of 6–7 h). When she moved, she radically changed her dietary habits, switching from a homemade plant-based diet, using local fresh products, to a high-calorie diet, consisting mainly of sodas and processed, canned, and preserved food. She reported having gained between 5 and 7 kg, in the first few weeks, which she later lost rapidly due to anorexia, nausea, and occasional vomiting.

She was initially referred to the local Centro de Salud Santa Maria Zacaltepec, where laboratory tests were performed and serum creatinine was found to be 3.6 mg/dL. She was then referred to the local hospital, and since she would not accept a therapeutic interruption of pregnancy, she was referred to INPER, for surveillance and, if necessary, dialysis start.

On referral to INPER, the physical examination revealed a woman who appeared to be in good health. She weighed 54 kg and was 148 cm tall (BMI: 24.7 kg/m²); her blood pressure was normal (132/74 mm Hg) on low-dose alpha-methyl-dopa, and she had no oedema, in spite of nephrotic-range proteinuria (Table 1). Kidney ultrasounds showed small poorly differentiated kidneys, suggesting the presence of a chronic interstitial disease (Fig. 1). Foetal ultrasounds detected a female foetus, normal for gestational age, with normal uteroplacental and umbilical flows. Given normal foetal growth and the absence of clinical indications for dialysis and also considering the economic and logistic burden of dialysis, we decided to attempt a nutritional approach first, consisting in a plant-based diet, supplemented with vitamins, calcium, and erythropoietin.

The patient's baseline protein intake, which was estimated at 1.2–1.3 g/kg/day, was mostly of animal origin (>70%), but the assessment may have been affected by the consumption of poor-quality food ("junk food"), whose protein content may be lower than expected.

Fig. 1. Kidney ultrasounds showing small poorly differentiated kidneys, in line with a chronic, long-standing disease.

In the proposed diet, protein intake was only slightly reduced (1.0 g/kg/day), but the source of proteins was changed (only 30% of animal origin), and the patient was also told to choose fresh fruits and vegetables and scheduled for strict checkups in the hospital (Table 2). Low-dose acetylsalicylate, folic acid, oral iron, and bicarbonate supplementation were started. One week later, laboratory findings had improved remarkably (Fig. 2; Table 1), with a striking reduction in BUN.

To be able to continue follow-up, the patient had moved to Mexico City with her husband, who had found a temporary job as a helper in a street food stall. They lived in a small room with shared access to a kitchen and had no refrigerator. Because of these economic and logistic constraints, at the subsequent control (28 weeks), while overall adhering to a plant-based diet, she had resumed a diet rich in packaged and junk food. Hypertension control was suboptimal. On account of the important logistic barriers, she accepted being hospitalized, so she would be able to follow the nutritional and treatment plan.

Throughout the following weeks, blood pressure remained well controlled on alpha-methyldopa (500 mg × 3 times per day). Serum creatinine remained overall stable; urea showed a trend towards a late increase, but remained lower than at referral (Table 1; Fig. 2). At 35 gestational weeks, weight gain was 4.1 kg since referral, and the diet was further modified, due to the onset of mild glucose intolerance. Foetal growth was normal throughout the pregnancy.

Caesarean section was planned at 37 weeks, on account of the presence of placenta accreta. A female baby weighing 2,460 g (11th centile for gestational age) was delivered, with an Apgar score of 7 at 1 min and 9 at 5

Table 1. Biochemical data during pregnancy

	25 weeks at referral, before dietary plan	26 weeks, 1st week on diet	28 weeks, partial dropout from diet	32 weeks, in hospital care	35 weeks, in hospital care	37 weeks, delivery	2 weeks after delivery
sCr, mg/dL	4	3.9	4.5	3.7	3.8	3.9	3.8
BUN, mg/dL	69.1	36.4	46.7	51.4	54.2	56.1	46.7
e-GFR CKD-EPI, mL/min	13.3	13.7	11.5	14.6	14.1	13.7	14.1
Serum albumin, g/dL	2.8	3	3	3.2	3.6	3.1	3.1
Total proteins, g/dL	6.5	6.4	6.6	6.3	6.3	6.2	6.2
Proteinuria, mg/day	3,500	3,230	3,560	1,694	2,168	2,496	3,376
Haemoglobin, g/dL	8.7	9.4	9.8	9.7	9.8	11.1	10.7
Calcium, mmol/L	9.0	9.1	9.1	8.8	9	9	8.4
Phosphate, mmol/L	5.0	4.7	5.0	4.4	4.2	4.8	4.3
Potassium, mmol/L	5.2	5.4	5.4	4.5	6.0	4.1	4.3
Uric acid, mg/dL	7.5	6.7	7.8	8.6	7.6	6.7	6.9
HCO ₃ , mEq/L	15	18	20	22	22	20	20
PTH, pg/mL	na	na	na	145.3	na	na	327
Vitamin D, pg/mL	na	na	na	45	na	na	50
Weight, kg	54	54	54.5	55.5	57	58.1	56
BP, mm Hg	136/80	125/80	138/81	110/70	100/70	116/69	126/72
Antihypertensive therapy	AMD 500 mg ×3	AMD 500 mg ×3	AMD 500 mg ×3 Niph 30 mg	AMD 500 mg ×3 Niph 30 mg	AMD 500 mg ×3 Niph 30 mg ×2	AMD 500 mg ×3 Niph 30 mg ×2	Niph 30 mg
Other treatments	ASA, folic acid	ASA, folic acid, Vit D, EPO	ASA, folic acid, Vit D, EPO	ASA, folic acid, Vit D, EPO	ASA, folic acid, Vit D, EPO, HCO ₃ iron, K binders	ASA, folic acid, Vit D, EPO, HCO ₃ iron, K binders	ASA, folic acid, Vit D, EPO, HCO ₃ iron, K binders, CaCO ₃

sCr, serum creatinine; GFR, glomerular filtration rate; PTH, parathyroid hormone; BP, blood pressure; AMD, alphamethyl dopa; Niph, niphedipine; ASA, acetylsalicylate; Vit D, 25-OH vitamin D; EPO, erythropoietin.

min. The baby was kept under observation in the neonatal intensive care unit on account of transient tachypnea of the newborn; the high bilirubin and uric acid levels detected responded to hydration and phototherapy. A small interventricular communication that was detected at birth spontaneously resolved after a few days; the baby was discharged in good clinical condition at 17 days of age.

The mother was discharged in stable clinical conditions 10 days after delivery (serum creatinine 3.84 mg/dL). At the first follow-up visit, 1 week later, she felt well, blood pressure was fully controlled on Nifedipine, and kidney function and proteinuria were stable.

Subsequent follow-up was planned in the hospital nearest to her home; a nutritional plan with a plant-based diet and a moderate protein restriction was counselled, while potential family donors were evaluated, to make it possible to plan a pre-emptive transplantation in case of further deterioration in kidney function.

Nutritional Management

Diagnosis: excessive proportion of proteins of animal origin (70%) related to poor choice and limited access and availability of fresh food.

Table 2. First dietary plan

	Servings	Kilocalories	Proteins	Lipids	Carbohydrates
Vegetables	2	50	4	0	8
Fruit	3	180	0	0	45
Fat-free cereals	11	770	22	0	165
Animal-derived food (low fat)	3	120	21	3	0
Whole milk	1	150	9	8	12
Oil without protein	4	180	0	20	0
Oil with protein	2	140	6	10	6
Overall		1,590 kcal	62 g	41 g	236 g

Distribution of protein intake: animal-derived proteins: 21 g/34%, plant-derived proteins: 41 g/66%.

Tortillas are not all the same

Recipe for homemade tortillas

(modified from <https://www.tasteofhome.com/recipes/homemade-tortillas> And <https://hungrydoug.com/recipe/worlds-best-flour-tortillas>)

Ingredients for 8 tortillas

- 2 cups all-purpose flour
- 1/2 teaspoon salt
- 3/4 cup water
- 3 tablespoons olive oil (or other fat, such as, traditionally, lard)

Nutrition Facts

1 tortilla: 160 calories, 5 g fat, 150 mg sodium, 24 g carbohydrates, 3 g protein.

Ingredients in industrially-produced tortillas

Different brands use different proportions and additives, with different attention to health-related issues:

(modified from: <https://www.tortilla-info.com/downloads/Europe%2018Tortilla%20Processing%20Parameters%20-%20Desiderio.pdf>)

- Wheat flour
- Water
- Salt
- Vegetable shortening
- Chemical leavening agents
- pH control & mould inhibitors
- Dough conditioners
- Reducing agents, emulsifiers, gums
- Sugar, enzymes, glycerin...

Fig. 2. Homemade and industrially produced tortillas.

Aims: to improve food choice, reducing animal-derived proteins (hamburgers, chicken, ready meals, etc.), returning to traditional habits, based on small quantities of different foods wrapped in tortillas.

The initial suggestions are reported in Table 2. Figure 1 reports the content of homemade and industrially produced tortillas.

Discussion

The Case

A plant-based diet is not a commercial “French fries diet,” meaning with this a diet based on processed and “fast food” or even “junk food” even if of plant origin. The quality of the diet matters.

Table 3. Main contaminants and their effect on health

Contaminant	Food	Toxic effect
Metals: Cadmium	Fish, vegetables, and cereals	Accumulates in the bones and liver; can cause cancer and reproductive system dysfunction; alters the development of the foetus in pregnancy
Lead		Accumulates in the bones and can damage the central nervous system, kidneys, and muscle system
Mercury		Negatively affects brain development in infants; can accumulate in the kidneys, liver, and central nervous system
Arsenic		Can cause skin lesions, cancer, neurotoxicity, cardiovascular diseases, abnormal glucose metabolism, and diabetes. There is emerging evidence of negative impacts on foetal and infant development, particularly reduced birth weight
Bisphenol A	Canned food	Can cause hormone disruption, reproductive disorders, and delayed menarche in females, infertility in males, and breast and prostate cancer
Phthalates	Food stored in plastic packaging	Exposure in women has been linked to disrupted thyroid hormone levels, increased systemic levels of oxidative stress inflammation, and breast cancer
Pesticides (i.e., organophosphates and their metabolites)	Fruit and vegetable	Have a neurotoxic effect; alter the development of the foetus in pregnancy
Parabens	Ubiquitous, used in food preservation	Have been hypothesized to have estrogenic properties and alter hormone metabolism to contribute to endocrine disruption
PFAS	Fish, vegetables, and cereals	Have been shown to interact with blood proteins; are thought to cause pathological responses and diseases, including cancer
N-nitroso compounds	Cured meat	Have carcinogenic potential as they can form adducts with DNA
Polycyclic aromatic hydrocarbons	Smoked meat	
Heterocyclic aromatic amines	Meat cooked at high temperatures	
PFAS, per- and polyfluorinated alkyl substances.		

The case discussed above is a good example of how pregnancy is an underexploited occasion for early diagnosis and follow-up of CKD and may show how pregnancy is an opportunity for reflecting on the importance of a nutritional approach that goes beyond considering quantity and type of proteins and involves attention to the quality of food.

Our patient was presumably diagnosed with CKD during her first pregnancy, yet she had only partially retained the information. In fact, while she had integrated the risks involved in a subsequent pregnancy, the diagnosis of CKD was not fully understood. In spite of the fact that CKD was long-standing and had gone untreated, the disease seemed clinically stable, as witnessed by her good clinical condition, well-controlled hypertension, and the lack of nephrotic syndrome at referral, thus making AKI on CKD unlikely (Fig. 1). Furthermore, her urea level was in keeping with the expected one in a patient on a mixed-protein diet and

stage 4-5 CKD, making severe hypercatabolism unlikely (Table 1).

In this setting, the nutritional interview highlighted not only a high percentage of animal-derived proteins but also, and possibly more importantly, their low quality. Notably, nutritional intervention did not substantially reduce protein intake, but switched it from poor-quality animal-based to higher-quality plant-based sources (Table 2); conversely, the most important difference between the patient's diet at home and in the hospital was the quality of food (cheap, preserved food at home, vs. mainly fresh food, in the hospital, in keeping with INPER policy).

While it is not possible to formally distinguish between the effects of the lower "kidney toxicity" of plant-derived proteins versus the avoidance of added toxic substances, this case is strikingly similar to another one recently published by our team, regarding a young Italian woman, affected by a complex anomaly of the kidney and urinary

Table 4. Main additives in food and their potential toxic effects

Category	E number (Europe)	Where found	Potential toxic effect
Dyes	E100–E180	Precooked or preserved food, sauces, powder dressings, nuts, preserved and processed meat, filled pasta (tortellini and ravioli), concentrated fruit juices, soft drinks, processed cheeses, wine, puddings, jellies, and candy	Carcinogenic potential; allergies; some are related to hyperactivity in children
Preservatives	E200–E297		High blood pressure, heart diseases, and cancer
Antioxidants and acidity regulators	E300–E385		Most are acceptable and even useful in certain cases, but high doses can worsen an existing disease. This is the case of phosphorus-based additives in kidney failure
Stabilizers, emulsifiers, and thickeners	E400–E585		Bowel distention and obstruction, malabsorption, mucous membrane irritation, and worsening of kidney and liver dysfunction
Flavour enhancers	E620–E640		Allergies and gastrointestinal dysfunction (diarrhoea, vomiting, and nausea)
Coating agents	E900–E948		Allergies and malabsorption of fat and fat-soluble vitamins
Sweeteners	E950–E967, E420, E421		Potential carcinogenic effect and allergies

tract [11]. This patient was also managed in pregnancy by changing her eating behaviour, without changing protein intake, switching from a moderately protein-restricted diet in which at least half of the calories derived from junk food to a plant-based diet, rich in fresh food and vegetables. These 2 cases, coming from differently resourced settings, were both characterized by a rapid decrease in urea level, together with a favourable trend, mainly involving creatinine in the Italian patient and phosphate in the Mexican one [11].

Our patient was also treated by other common drugs in advanced CKD: for instance, erythropoietin, vitamin D, and bicarbonate were added to treatment, and blood pressure control was optimized. However, none of these is effective in the short term on urea levels, and we considered that the metabolic effects observed were presumably due to the diet changes.

In both cases, the biochemical trends are not explained merely by the change in protein source, but suggest that the dietary changes had led to the avoidance of substances, such as polyphosphates or additives, that may exert a direct effect on kidney function. Notably, in both cases, in spite of advanced CKD, and in keeping with the importance of nutritional management, the babies were normal for gestational age and were born late preterm (the Italian case) or at term (the Mexican case).

Diet Quality and Additives

A poor-quality diet is a source of substances that are potentially harmful to health. We can distinguish between substances that are intentionally added to food, such as additives, or preservation agents, and substances that are not intentionally added to food but result from chemical changes during food production, processing, packaging, or transport or come from environmental contamination (Tables 3, 4). Both categories contain substances that are potentially harmful but the information we have is limited, for several reasons: some preservation agents, including phosphates and potassium, are not declared on food labels; interest in these agents is recent, and studies on humans are lacking. In animal studies, these compounds are often administered in higher doses and for short periods of time. Furthermore, in processed food, several additives are present at the same time, and disentangling the effect of each one may be difficult.

The main reasons why additives are added to food are to guarantee microbiological safety, extend shelf life, and maintain organoleptic characteristics. They are the consequence of the industrial production and distribution cycles: most of the food products in our everyday diet contain different amounts of additives of various chemical origins. In the USA, food additives must be authorized by the Food and Drug Administration (FDA), and in Europe, they must be authorized by the European Food Safety Author-

ity (EFSA). The market has grown very rapidly recently, and in spite of the protective legal measures, concerns exist about their long-term safety [29–31]. By law, producers must declare the presence of additives on food labels, but are not obliged to indicate the amounts contained; their presence is often indicated with the letter “E” followed by a number that specifies a class (e.g., dyes, antioxidants, thickeners, emulsifiers, and preservatives) [32, 33].

Different adverse reactions, including, but not limited to, eosinophilic esophagitis, bronchial asthma, atopic dermatitis, contact dermatitis, chronic urticaria, anaphylaxis, metabolic syndrome, and inflammatory and bowel diseases, have been described [29, 30]. Patients with reduced kidney function appear to be particularly prone to developing toxicity from many kinds of chemicals. However, data are scant and mainly regard phosphates; thus, the advantages of an additive-free diet, if one were actually possible, are not clear [31, 34–36].

Even less is known about contaminants unintentionally added to food or substances from polluted marine and agricultural food chains. Examples are the curing of meat with nitrite, which can produce *N*-nitroso compounds, and the smoking of meat which produces polycyclic aromatic hydrocarbons, while cooking at a high temperature produces heterocyclic aromatic amines: these chemicals have a carcinogenic potential [37–40]. Consumption of processed meat is associated with higher risk of chronic heart disease and diabetes mellitus [40, 41]. Seafood can contain significant amounts of mercury and arsenic, which are actively bio-accumulated; concentrations depend on the species, habitat, maturity, weight, and life span [42–44].

High cadmium concentration has been found in soil irrigated with wastewaters [45, 46]. The kidney is the main organ affected by chronic cadmium exposure, and toxicity leads to a range of disorders from tubular proteinuria to renal failure [47, 48].

The list is long and still incomplete: for example, consumption of canned food has been related to increased urinary levels of bisphenol A, a synthetic chemical with various adverse effects on health, used in the production of polycarbonates in digital media (CDs and DVDs), sealants used by dentists, electronic equipment, tableware, and reusable bottles [49]. Its health hazards are linked to estrogenic activity that can lead to reproductive disorders, delayed menarche in females, infertility in males, and breast and prostate cancer [50]. Other adverse effects of early exposure are neurodevelopmental impairment with various manifestations, including attention deficiency, autism, and hyperactivity [51].

Consumption of food stored in plastic packaging leads to higher urinary levels of phthalate and its metabolites [52]. Phthalates are almost ubiquitous: 2–5 di (2-ethyl-hexyl)phthalate exposure occurs when contaminated food or water are consumed, while exposure to a number of phthalates (e.g., benzylbutyl phthalate or diethyl phthalate) occurs through contact with perfumes and deodorants. Exposure has been linked to thyroid derangements, increased oxidative stress, inflammation, and breast cancer [53, 54].

Conversely, fruits and vegetables have been identified as a major source of organophosphates, compounds widely used as pesticides, as they degrade easily under natural condition; however, content may still be high in ripe fruit. Residues have been detected in water, soil, and in products such as tea, sugar, vegetables, and fruits [55]. Parabens are antioxidants used in the preservation of foods, to extend their sell-by dates, and in the production of cosmetics and drugs, thanks to their antibacterial and antifungal properties. They are almost ubiquitous and have been found in soil, wastewater, rivers, and house dust; like phthalates, parabens may have estrogenic properties [56, 57].

Per- and polyfluorinated alkyl substances are another group of compounds eliciting increasing interest. They are used in manufacturing fabrics, paper, and food containers and in the production of photographic films, fire-fighting foams, and household detergents. Recent studies have shown that the wastewater treatment system is also a significant source of perfluoroalkyl compounds, potentially cancerogenic. Plants can absorb these compounds, whose uptake varies depending on species, chain length, and functional group. For example, accumulation of perfluorooctanoic acid and perfluorooctane sulfonic acid has been found in potatoes and grains, while high levels of short-chain compounds have been found in fruits and green vegetables, fish, and seafood [58, 59].

While data on patients with chronic kidney disease are scant, the long list of contaminants discussed here should lead us to reflect on their potential negative effect on kidney function and make us realize that eating healthy, unprocessed “organic” food should be encouraged, in particular in delicate conditions such as pregnancy and breastfeeding and for young children, in particular when kidney function is failing. These nutritional aspects may add to the long list of elements to which a multidisciplinary team, fundamental in the management of high-risk pregnancies, has to pay attention in the care of patients with advanced CKD in pregnancy.

Conclusions

This is a review based on a “one-case trial,” and we have to be cautious in drawing generalized conclusions. However, the suggestions originating from the case history support an effort to modify unhealthy dietary habits, in patients with advanced CKD, in particular if pregnant. In this context, our review suggests that it is vital to focus attention not only on macronutrients (proteins vs. others) and on their sources (animal vs. plant based) but also on the overall quality of the food eaten (fresh vs. preserved), to try to improve overall and kidney health in CKD patients.

Statement of Ethics

The patient has given written informed consent to publish her case (including publication of images).

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Author Contributions

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