

**Georgia State University
Dietetic Internship Program
Evaluation of Written Assignments – Case Study**

Name Nupur Date 6/24 Topic CKD
 Evaluator Jessica Johnson

	Comments
Preparation (10 points) Appearance is neat; assignment is legible; scheduled deadline met	10
Introduction (5 points) Provided a brief introduction to the topic	5
Content (50 points) Organized the information into appropriate sub-topics. Summarized the relevant research; identified the relationship and understanding between the existing knowledge, strengths and limitations of the current knowledge; comprehensive coverage of subject. The literature review was comprehensive and up-to-date. All components of the Nutrition Care Process were included.	<ul style="list-style-type: none"> • Didn't discuss strengths, limitations. • Did not include Vitamin D into the lit review even though it was part of the labs. This could have been a change you could have made to the intervention. 42
Style (25 points) Assignment was organized; correct spelling and word usage; appropriate sentence structure; proper use of punctuation	<ul style="list-style-type: none"> • used first person • spelling 18 + silly mistakes
Bibliography: (10 points) All were references were listed and complete information was provided. The reference citation format was identified on the reference page. (JADA, JAMA, or Am J Clin Nutr may be used).	10
Overall	85
Total Points _____ /100 pts	

NUPUR VIDWANS

Introduction

Mrs. C is a 46 year old African American Female with a history of hypertension, hyperlipidemia and CKD stage 4. She came into the hospital with chief complaint of anasarca (generalized swelling of body due to fluid retention) on May 1st 2013. According to her medical record she was a candidate for Peritoneal Dialysis and had an appointment for vascular port insertion on may 7th, but due to sudden onset of fluid accumulation she was admitted a week prior. Mrs. C is 5'4" in height and weighs 185 pounds but her admission weight was 205 pounds due to fluid accumulation, her BMI is 32 kg/m² (dry body weight). She is a school canteen staff and normally has her lunch at the school cafeteria. She said that her typical diet is ~~some~~ cereal and milk (low fat) for breakfast, which she eats in car on way to school. Her lunch is at school, which is usually at 12:30 pm ^{and} consists of a pizza slice, bread with some meat and fruit (basically whatever is served at school). Her dinner is a piece of meat some vegetables and fruit. She also reported that prior to the diagnosis of CKD she used to eat very unhealthy (processed meats) but since the diagnosis she changed her eating habits. She also ~~informed~~ ^{reported} that she read a lot about CKD and according to ~~that~~ ^{those} readings she had completely stopped ~~including~~ ^{adding} salt to her meals. She restricted her fluids to 8 cups/day. ~~As~~ ^{even though} she had some knowledge on CKD, she was eager to know more and from a professional source.

Mrs. C is currently on Renavite, Cepacol, Coreg, Colace, Lasix and was recently started on Coumadin. Upon admission she was placed on renal diet.

Nutrition was consulted for education on renal diet and Coumadin education (drug-nutrient interaction).

Her lab reports:

Date	5/1	5/2	Normal
HbA1C	4.9		
Na meq/l	139	138	135-145
K meq/l	3.7	4.1	3.5-5.5
Cl meq/l	117	111	95-105
Glucose mg/dl	72	105	70-130
BUN mg/dl	31 ↑	34 ↑	6-20
Creat mg/dl	3.9 ↑	4.0 ↑	0.8-1
GFR	15 ↓	15 ↓	
Albumin gm/dl	<1.5 ↓	<1.5 ↓	3.5-5.5
P meq/dl	5.8 ↑	5.4 ↑	2.4-4.1
Vit D U/L	10 ↓		
Cholesterol mg/dl	449 ↑		
HDL	55		>50
LDL	374 ↑		< 100

Interpretation of lab values:

Since Mrs C was a CKD stage 4 pt, it clearly depicted in her lab values. Her BUN, Creat and phosphorus are high and she has a low GFR. This happens to pt with kidney disease as the kidneys are unable to filter out the creat and nitrogen and as a result it starts building up in body, which is, can be inferred through the serum values. Her serum lipid values are high as she is a known case of hyperlipidemia (from her case report).

What about the low albumin? (Fluid overload?) Vitamin D?

PES statement;**Nutrition diagnosis:**

- 1: food and nutrition related knowledge deficit related to CKD as evidenced by conversation with pt.
- 2: food-medication interaction (vit K) ~~as~~ related to Coumadin as evidenced by her medical records.

Interventions: nutrition relationship to health and diseases.

Monitor: Monitor the pt for phos, BUN and creat levels

Evaluation: verbalize understanding prior to discharge.

Plan:

- 1: the pt diet was changed to 60 gm protein renal diet.
- 2: educated the pt on Coumadin and Vit K
- 3: educate the pt on diet and CKD

Lit Review of significant nutrition related issues

Chronic kidney disease (CKD), also known as chronic renal disease, is a progressive loss in renal function over a period of months or years. The symptoms of worsening kidney function are non-specific, and might include feeling generally unwell and experiencing a reduced appetite. Chronic kidney disease may also be identified when it leads to one of its recognized complications, such as cardiovascular disease, anemia, or pericarditis. Chronic kidney disease is identified by a blood test of creatinine and BUN levels. Higher levels of creatinine indicate a lower glomerular filtration rate and as a result a decreased capability of the kidneys to excrete waste products. The most common causes of CKD are diabetes, hypertension, and glomerulonephritis. Mrs. C had a known history of HTN and glomerulonephritis.

Vit K and Coumadin:

Vitamin K in its reduced form is the essential cofactor for post translational activation of the vitamin K dependent clotting factors, the procoagulants – factors II, VII, IX, X, and the anticoagulant proteins C and S. In the reaction, glutamic acid is converted to γ -carboxy-glutamic acid by γ -glutamyl-carboxylase, and vitamin K1 is converted to vitamin K epoxide which is rapidly reduced back to vitamin K quinone by the Vitamin K Epoxide Reductase Complex 1 (VKORC1) and then to vitamin K hydroquinone (KH₂). VKORC1 is the molecular target inhibited by warfarin, which exerts its anticoagulant activity by interrupting the regeneration of KH₂, the active (reduced) form of vitamin K, leading to decreased carboxylation of the vitamin K dependent clotting factors with loss of activity. (1,2). Since Mrs. C was on Coumadin she has to limit her Vit K intake, as it will interfere with the action of Coumadin. Hence she was educated and a handout was provided to Mrs. C on how to limit or regularize her Vit K intake.

Energy needs of CKD Stage 4:

The energy requirement of patients with chronic kidney disease (CKD) is increased as patients with CKD have symptoms of anorexia and consequently results in reduced food intake. However, data regarding energy expenditures of CKD patients are still scarce, and the results obtained are conflicting, with studies showing energy expenditures to be similar, higher, or lower than those of healthy individuals (3).

The pt is currently diagnosed and staged as CKD stage 4 and was not started on dialysis. Hence referring to the AND care manual she was provided 30 kcals/kg RBW/day (AND nutrition care manual)

What was her recommended body weight and how many calories did this equate to? What about protein?

Fluid and sodium restriction

Sodium and intravascular volume balance are usually well maintained until the GFR falls below 15 mL/min/1.73 m². This is caused by an increase in the fractional excretion of salt and water by the remaining nephrons. However, the ability to respond to rapid infusions of sodium with volume expansion will be reduced, even in patients with CKD stages 3 and 4, making them prone to fluid overload. The optimal level of daily salt intake varies from patient to patient. Less than 6 g/day of sodium chloride (<2 g/day of sodium) is the typical initial recommendation. Adjustments need to be made depending on the patient's volume status, aiming to achieve normotension. Patients with a GFR below 20 mL/min/1.73 m² in whom, despite sodium restriction, edema ensues, respond well to diuretic therapy, usually a loop diuretic. Given that the ability to concentrate or dilute the urine maximally becomes progressively impaired as GFR declines, patients with stage 4 or 5 CKD tend to be isosthenuric (Isosthenuria refers to the excretion of urine whose specific gravity is neither greater nor less than that of protein-free plasma, typically 1.008-1.012). Therefore, these patients are at risk for developing hypo- or hypernatremia caused by positive or negative water balance, respectively. Free water

intake should be approximately equal to urine output plus an additional 1 to 1.5 L/day to account for insensible losses. (4,5,6). Though Mrs. C was a stage 4 CKD pt she was not on any fluid restriction (GFR 15) and not fallen below that level, though her sodium was limited to 2 gm per day.

Protein requirement:

The Modification of Diet in Renal Disease (MDRD) study was the largest controlled multicenter trial to compare usual protein intake (1 g/kg/day) with low (0.6 g/kg/day) and very low (0.28 g/kg/day) protein intake in nondiabetic patients. Although the primary outcome was inconclusive, several subanalyses have suggested that a prescribed dietary protein intake of 0.6 g/kg/day as compared with 1 g/kg/day reduces the rate of progression of kidney disease by about 28%, the same benefit seen in achieving the low blood pressure goal. A meta-analysis of five studies of both diabetic and nondiabetic renal disease has suggested that a small reduction in rate of progression occurs with dietary protein restriction. In an analysis of the MDRD data, Locatelli and Del Vecchio ~~have~~ found that adherence to a low (0.6 g/kg/day) versus a usual (1 g/kg/d) protein diet for 9 years would delay the need for renal replacement therapy by approximately 1 year. The difficulty of achieving consistent dietary protein restriction, however, makes the application of this intervention unwieldy and prone to failure. (7, 8)

Phosphorus:

As functional nephron mass declines, the fractional excretion of phosphate drops, leading to an increase in the serum phosphate level. This is accompanied by a reciprocal decrease in serum calcium concentration. These events lead to an increase in parathyroid hormone (PTH) release; this has a phosphaturic effect, resulting in the return of phosphate and calcium to normal levels. As GFR continues to decline, this cycle maintains serum calcium and phosphate concentrations within the normal ranges, at the expense of rising PTH levels. When further renal mass is lost and GFR drops below 30 mL/min/1.73 m², despite the compensatory hyperphosphaturia, hyperphosphatemia is seen normally in patients. The normal phosphorus intake recommended varies from 600-800 mg/day in order to prevent hyperphosphatemia. (9,10)

Recommendations for Mrs. C:

The diet was planned for Mrs. C based on the below mentioned macro and micronutrient levels. Though the above-mentioned literature review was not done at that time, as the consult was supposed to be completed in 24 hrs, the recommendation were based on AND care manual, which was available in the hospital.

Protein: 0.6-0.8gm/kg/day

Na: < 2gm/day

Phosphorus: 600-800 mg/day

Currently the pt was not on any fluid restriction.

Based on these guidelines the diet was planned and pt was educated on this components.

Hence Mrs. C's diet was planned providing 1740-2000 kcals, (30-35 kcals/kg) 35-43 ~~gm~~ grams of proteins per day and level was maintained between 600-800 mg/day.

— what level? Phosphorus?

Mrs. C was very eager to know about the diet and was very receptive to life style changes. Through research Mrs. C had ~~found out~~ ^{learned} many things pertaining to diet and CKD and during the education session, ~~she clarified them~~ ^{clarified them?}. The education was done both orally and with handouts on salty do's and don'ts. The other handouts included ways to reduce Na, K and phos in diet. She was given a meal plan that met her needs for proteins, kcals, and phos. In addition a handout was provided on diet in CKD (providing an overview of food choices, prot and Na and phos content of various foods) and vit K and Coumadin education. Though she was hyperlipidemic, this issue was not addressed ~~at this time~~ ^{during the consult} as

CKD was given a priority. Since ~~it~~ ^{she} was to be ~~strated~~ ^{start} on dialysis soon Mrs. C was made aware that her diet plan would change once she ~~begins~~ ^{started} dialysis and advised her ~~for~~ ^{she was} follow up. Follow up was completed before discharge and ~~pt~~ ^{she} reported to have understood the provided education and was ready to make life style change, to achieve better outcomes.

Since the literature review was done after the diet prescription, going over the literature according to me there will not be any changes in my previous recommendations. The ADA care manual is based on the literature and the above mentioned literature review just supports the manual too.

References:

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2. Garcia AA, Reitsma PH. VKORC1 and the vitamin K cycle. Vitam Horm 2008; 78:23-33.
3. Carrero, J.J.; Qureshi, A.R.; Axelsson, J.; Avesani, C.M.; Suliman, M.E.; Kato, S.; Bárány, P.; Snaedal-Jonsdottir, S.; Alvestrand, A.; Heimbürger, O.; et al. Comparison of nutritional and inflammatory markers in dialysis patients with reduced appetite. Am. J. Clin. Nutr 2007; 85:695-701.
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5. Sanders PW. Dietary salt intake, salt sensitivity, and cardiovascular health. Hypertension 2009;53:442-445.

Diet prescription was based on the AND Care Manual which is based upon the literature

Even after completing the literature review, no changes to the intervention should have been made

Spell out

Spell out

First person

6. Emma J. McMahon, Katrina L. Campbell, David W. Mudge, and Judith D. Bauer. Achieving Salt Restriction in Chronic Kidney Disease. *Intl Jr of Nephrol.* 2012 Article ID 720429, 10
7. Kalantar-Zadeh K, Cano NJ, Budde K, Chazot C, Kovesdy CP, Mak RH, et al. Diets and enteral supplements for improving outcomes in chronic kidney disease. *Nat Rev Nephrol.* 2011; 7:369–84.
8. Bellizzi V, Di Iorio BR, De Nicola L, et al. Very low protein diet supplemented with ketoanalogues improves blood pressure control in chronic kidney disease. *Kidney Int* 2007;71(3):245–51.
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