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Estimation of Protein Intake on the Basis of Urinary Urea Nitrogen in Patients with Non Alcoholic Fatty Liver

Sneha Susan Mathew¹, Lakhvinder Kaur²

^{1,2}Department of Nutrition & Dietetics, Manav Rachna International Institute of Research and Studies, Faridabad, Haryana-121004

Abstract: Purpose The present study has been undertaken to determine effectiveness of the estimated protein intake (EPI) based on urinary urea nitrogen (UUN) in patients with NAFL

Methods A total of 50 patients with NAFL were taken into consideration in this study. The actual protein intake (API) of the patient was determined through 7 day dietary recall. The estimated protein intake (EPI) was calculated using the following equation: $EPI = [UUN (g/d) + 0.031 \times \text{body weight (kg)}] \times 6.25$

Results The correlation between API and EPI was examined. As compared to API, EPI was found out to be lower due to low bioavailability of protein sources in the diet and therefore the correlation between API and EPI was non significant as $p > 0.01$.

Conclusion It can be concluded that UUN is one of the novel methods in order to find out EPI if all the limiting factors can be taken care of. Also this study needs further consideration to find out much more accurate correlation between UUN and EPI.

Keywords: Urinary urea nitrogen, Non alcoholic fatty liver disease, actual protein intake, estimated protein intake,

I. INTRODUCTION

Protein regenerates hepatocytes and provides necessary amino acids that inhibit more of fat build up within hepatocytes. High protein intake has associations with higher catabolic rate of amino acids. This leads to high energy expenditure and lipid oxidation in liver. Previous clinical trials showed that protein intake has a beneficial impact on NAFLD patients with a fair increase in protein intake (15.4% of total calorie intake) combined with low-GI diet. The recommendation of protein is 0.8g/kg ideal body weight/day according to American physiological society. Optimal nutritional support is provided through protein estimation of patients with non alcoholic fatty liver, however UUN has never been used in these patients. On contrary, it was found to be useful in the estimation of protein intake in patients with early stage liver cirrhosis and chronic liver failure.

Evaluation of protein intake on the basis of UUN in patients with NAFL has never been employed in India and therefore it is the first study to validate protein estimation in non alcoholic fatty liver patients.

II. METHODOLOGY

Subjects with non-alcoholic fatty liver from private clinics and hospitals of Faridabad were taken into consideration in this study. 50 subjects were a part of the study. All subjects agreed to be a part of the study by signing written informed consent form. With the help of interview the etiological conditions of non-alcoholic fatty liver disease were identified. The subjects aged 30 to 40 years (averaged 35.06 ± 3.26), consisted of 25 males and 25 females. 68% of subjects responded to obesity condition whereas 52% of subjects responded to high blood pressure condition, 30% of subjects responded to hypercholesterolemia condition and only 24% of subjects responded to diabetes condition. Body mass index of the subjects were calculated (averaged 27.91 ± 2.48) and 72% of the subjects were found to be in overweight category whereas 22% of the subjects were found to be in obese class I category, 4% of the subjects were found to be in normal weight category and only 2% of the subjects were found to be in obese class II category. The UUN was found to be 0.84 ± 0.32 g/day. The actual protein intake (API) was determined through 7 day dietary recall which took into account energy (in kilo-calories), protein (in grams), fat (in grams) and carbohydrate (in grams). The estimated protein intake (EPI) was determined through 24 hour urinary urea nitrogen and the following equation was used to calculate it: $EPI = [UUN (g/d) + 0.031 \times \text{body weight (kg)}] \times 6.25$. The correlation between API and EPI was examined. The results shown were mean $\pm$ standard deviation (SD). Pearson correlation was to see the relationship between the variables.

III. RESULTS

Table I - Comparison of Etiological Conditions in Males and Females

Etiology	Male	Female	p value
Hypercholesterolemia	7(28%)	8(32%)	0.577
Obesity	17(68%)	17(68%)	0.827
Diabetes	4(16%)	8(32%)	0.313
High blood pressure	15(60%)	11(44%)	0.555

Table 1 shows the etiological conditions in males and females. 68% of males and 68% of females responded to obesity condition whereas 60% of males and 44% of females responded to high blood pressure condition, 28% of males and 32% females responded to hypercholesterolemia condition and only 16% of males and 32% of females responded to diabetes condition. $p > 0.05$, therefore there was no significant difference in the etiological conditions of males and females.

Table II- Mean Values of Dietary Components from 7 Day Dietary Recall in Males and Females

Variable	Gender	mean $\pm$ stddev	p value
BMI	Males	28.4 $\pm$ 2.4	0.167
	Females	27.5 $\pm$ 2.5	
Energy	Males	1313.0 $\pm$ 298.8	0.548
	Females	1253.7 $\pm$ 379.8	
Protein	Males	0.6 $\pm$ 0.2	0.712
	Females	0.6 $\pm$ 0.2	
Fat	Males	28.6 $\pm$ 10.5	0.541
	Females	26.5 $\pm$ 13.1	
CHO	Males	228.0 $\pm$ 43.7	0.184
	Females	208.3 $\pm$ 57.2	

Table II shows $p > 0.05$, therefore there was no significant difference in the BMI and dietary components from 7 day dietary recall of males and females.

Table III -Correlation Between BMI and Protein Among Subjects

Variable	mean $\pm$ stddev	p value
BMI	27.91 $\pm$ 2.48	0.971
Protein	0.64 $\pm$ 0.19	

Table III shows that $p > 0.05$, therefore there was no significant correlation between BMI and protein from 7 day dietary recall of males and females.

Table IV- Correlation Between Estimated Protein Intake and Urinary Urea Nitrogen

Variable	mean $\pm$ stddev	p value
UUN	0.84 $\pm$ 0.32	0
EPI	0.26 $\pm$ 0.03	

Table IV shows Urinary urea nitrogen was found to be 0.84 $\pm$ 0.32 and estimated protein intake based on urinary urea nitrogen was found to be 0.26 $\pm$ 0.03 among subjects. The correlation between urinary urea nitrogen and estimated protein intake was found to be significant as $p < 0.01$

Table V Correlation Between Actual Protein Intake and Estimated Protein Intake Among Subjects

Variable	mean $\pm$ stddev	p value
API	0.64 $\pm$ 0.19	0.918
EPI	0.26 $\pm$ 0.03	

Table V shows that actual protein intake based on 7 day dietary recall was found to be 0.64 ± 0.19 and estimated protein intake based on urinary urea nitrogen was found to be 0.26 ± 0.03 . The correlation between actual protein intake and estimated protein intake was found to be non-significant as $p > 0.01$.

IV. DISCUSSION

It has been proved that EPI can be useful tool for early stage liver cirrhosis and chronic renal failure. This study is first to prove that EPI can be calculated from UUN in patients with non alcoholic fatty liver disease. API was determined through 7 day dietary recall in patients. EPI was calculated by using the following equation: $EPI = [UUN (g/d) + 0.031 \times \text{body weight (kg)}] \times 6.25$. It has been found from the results that the main etiological factor behind NAFL was obesity as responded by patients followed by high blood pressure, hypercholesterolemia and diabetes. Obesity contributes to non alcoholic fatty liver occurrence as the rate of liberation of FFA from adipose tissue and transport to the liver and skeletal muscle is elevated in obese subjects. This ultimately increases FFA uptake in muscle and liver. Further there is an increase in the de novo lipogenesis in NAFLD subjects which leads to the build up of intracellular fatty acids. There appears a negative correlation between BMI and protein. Improper recording of dietary intake is the first limitation over here. A comprehensive link between dietary protein and individual components (i.e., fat, muscle, and bone) of body composition could not be assessed and therefore it appears to be the second limiting factor for us. The patient interviews and recordings of their dietary intake over 7 days was the method used to estimate quantity of protein intake in the subjects. The dietary recommendation of protein intake in NAFLD is 0.8-1g/kg body weight/day and the actual protein intake (API) came out to be 0.6 g/kg body weight/day in both males and females. The API was lower as the patients belonged to the low income category and their diet was not found to be rich in protein. In the study earlier conducted on estimation of protein intake in patients with early stage liver cirrhosis, API was considered to be the supplements containing BCAA and total protein intake from hospital meals (1.0g/kg/d). However in this study there was no fixed diet and supplementation due to which API came out to be lower. Though urinary excretion ensures nitrogen balance both in healthy individuals and in liver diseased patients, the protein catabolism of the body was found to have no good correlation with UUN and theoretically correlation would be significant only if the nitrogen balance is stable. The nitrogen balance is stable when nitrogen intake is equal to nitrogen loss. Nitrogen intake is calculated from division of the dietary intake of protein by 6.25 and nitrogen loss is calculated by addition of 4 to UUN which accounts for non urinary losses of nitrogen. $\text{Nitrogen balance} = \text{nitrogen intake} - \text{nitrogen loss}$. It was a limiting factor for us that we could not calculate EPI/API (EAR) ratio and corrected EPI based on EAR results. $\text{Corrected EPI} = \{ [UUN (g/day) + 0.031 \times \text{body weight (kg)}] \times 6.25 + \text{urine protein (g/day)} \} \times 1.2$ (because $1/0.84 = 1.19$), where $1/0.84$ is the adjustment of EAR to 1 in the research conducted before on early stage liver cirrhosis subjects.

Unavailability of the records of the patients containing urine protein tests and non willingness of patients to get the tests done due to their financial problems was the major limiting factor for the study. In general, 80% of urea nitrogen is excreted in urine. The non urinary urea nitrogen losses (the nitrogen in feces, in urinary creatinine, uric acid, peptides and all other forms of nitrogen except urea) was included in the formula for calculating estimated protein intake (EPI) and the average NUN value was 0.031g nitrogen/kg body weight/day. Thereby, since UUN was lower (0.84 ± 0.32 g/24 hour) than the normal range (6.0-20.0 g/24 hour), EPI also came out to be lower. One more limiting factor found out to be was the single measurement of UUN whereas it is recommended to measure UUN on 24 hour basis because of non availability of patients and their non willingness. As compared to API, EPI was found out to be lower due to low bioavailability of protein sources in the diet and therefore the correlation between API and EPI was found to be non significant.

V. CONCLUSION

On the basis of research discussion, it can be concluded that UUN is one of the novel methods in order to find out EPI if all the limiting factors mentioned above can be taken care of in the future research. Also this study needs further consideration to find out more accurate correlation between UUN and EPI.

REFERENCES

- [1] Yohei Kida, Hiroki Ueda, Hiroto Tanaka, Masakazu Ichinose, Estimation of protein intake using urinary urea nitrogen in patients with early-stage liver cirrhosis Hepatol Int (2007) 1:382-386
- [2] Alberino F, Gatta A, Amodio P, et al. Nutrition and survival in patients with liver cirrhosis. Nutrition 2001; 17:445-50.
- [3] Tajika M, Kato M, Mohri H, et al. Prognostic value of energy metabolism in patients with liver cirrhosis. Nutrition 2002; 18:229-34.
- [4] Moriwaki H, Miwa Y, Tajika M, et al. Branched-chain amino acids as a protein- and energy-source in liver cirrhosis. Biochem Biophys Res Commun 2004; 313:405-9.
- [5] Selberg O, Bottcher J, Tusch G, et al. Identification of high- and low-risk patients before liver transplantation: a prospective cohort study of nutritional and metabolic in 150 patients. Hepatology 1997; 25:652-7.

- [6] Plauth M, Carbe ´E, Riggio O, et al. ESPEN guidelines on enteral nutrition: liver disease. Clin Nutr 2006;25:285–94.
- [7] Friedman LS. Cirrhosis liver, biliary tract, & pancreas. In: Tierney LM, McPhee SJ, Paradakis MA, editors. 2006 Current medical diagnosis & treatment. 45th ed. New York: McGraw-Hill; 2005. p. 668–74.
- [8] McCullough AJ. The clinical features, diagnosis and natural history of nonalcoholic fatty liver disease. Clin Liver Dis. 2004;8
- [9] Angulo P. Nonalcoholic fatty liver disease. N Engl J Med. 2002;346(16):1221–31.
- [10] de Alwis NM, Day CP. Non-alcoholic fatty liver disease: the mist gradually clears. J Hepatol. 2008;48 Suppl 1:S104–12.
- [11] Bedogni G, Miglioli L, Masutti F, Tiribelli C, Marchesini G, Bellentani S. Prevalence of and risk factors for nonalcoholic fatty liver disease: the Dionysos nutrition and liver study. Hepatology. 2005;42(1):44–52.
- [12] Paola Dongiovanni, Claudia Lanti, Patrizia Riso, Luca Valenti Nutritional therapy for nonalcoholic fatty liver disease; Internal Medicine and Metabolic Diseases, Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico, 20122 Milano, Italy Department of Food, Environmental and Nutritional Sciences (DeFENS), Division of Human Nutrition, Università degli Studi di Milano, 20133 Milano, Italy Department of Pathophysiology and Transplantation (DEPT), Università degli Studi di Milano, 20122 Milano, Italy Journal of Nutritional Biochemistry 29 (2016) 1 – 11
- [13] Angulo P. Nonalcoholic fatty liver disease. N Engl J Med 2002;346:1221–31.
- [14] Browning JD, Szczepaniak LS, Dobbins R, Nuremberg P, Horton JD, Cohen JC, et al. Prevalence of hepatic steatosis in an urban population in the United States: impact of ethnicity. Hepatology 2004;40:1387–95.
- [15] Blachier M, Leleu H, Peck-Radosavljevic M, Valla DC, Roudot-Thoraval F. The burden of liver disease in Europe: a review of available epidemiological data. J Hepatol 2013;58:593–608.
- [16] Marchesini G, Brizi M, Morselli-Labate AM, Bianchi G, Bugianesi E, McCullough AJ, et al. Association of nonalcoholic fatty liver disease with insulin resistance. Am J Med 1999;107:450–5.

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