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IMPACT OF BALANCING GRAMS OF QUALITY PROTEIN INTAKE ON NUTRITIONAL STATUS AND QUALITY OF LIFE IN CKD PATIENTS

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Type& amount of protein to be ingested by CKD patients in order to keep the kidneys not to deteriorate further especially in Indian population who consume mostly vegetarian diet makes planning of diet based on protein quality ratio a tough job.
Objective: To analyze effect of optimizing the protein quality intake [high biological value (HBV), net protein utilization (NPU) or protein efficiency ratio (PER) of food article] on uremic toxins, nutritional status and quality of life in CKD patients consuming 0.6-0.8g/kg body wt. protein; 80% of which is from poor quality.
Method: 145 predialysis CKD patients were enrolled who completed a food frequency questionnaire, quality of life (QOL) performa, nutritional status evaluation (dietary intake, anthropometry, serum albumin, total protein) before and after diet counseling [$\geq$ 50%of HBV protein (from casein and egg base)]; energy 35-40 Kcal/kg body wt.).
Results: Creatinine reduced significantly ($p \leq 0.001$), non significant change in blood urea& GFR improved ($p \leq 0.01$).Nutritional intake increased[good quality proteins intake ratio ($p \leq 0.001$), energy($p \leq 0.001$)] ,BMI elevated ($p \leq 0.01$)and perception of QOL improved after diet counseling
Conclusion: Judicious planning of quality protein intake within restricted quantity along with calorie optimization is critical to reduce protein waste products. Therefore, proper &timely diet counseling to combat ignorance & impart awareness to CKD patients is of utmost importance.

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EFFECTS OF L-CARNITINE SUPPLEMENT ON PLASMA COAGULATION AND ANTICOAGULATION FACTORS IN HEMODIALYSIS PATIENTS

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Background: Hypercoagulability is an important risk factor for thrombosis and its complications in hemodialysis patients. This study was designed to investigate the effects of L-carnitine supplement on plasma coagulation and anticoagulation factors in hemodialysis patients.
Methods: Thirty-six hemodialysis patients were randomly assigned to either a carnitine or a placebo group. Patients in the carnitine group received 1000 mg/day oral L-carnitine for12 weeks, whereas patients in the placebo group received a corresponding placebo. At baseline and the end of week12, 5 mL blood was collected after a 12- to 14-hour fast and plasma fibrinogen concentration, activity of plasma proteinC, coagulation factors V, VII, IX, and serum concentrations of tissue plasminogen activator (tPA), plasminogen activator inhibitor type-1 (PAI-1), free carnitine, and C-reactive protein (CRP) were measured. Results: In the carnitine group, mean serum free carnitine concentration increased significantly to 150% of baseline ($p < 0.001$), whereas plasma fibrinogen and serum CRP had 98 mg/dL ($p < 0.01$) and 41% ($p < 0.01$) significant decreases, respectively, at the end of week 12 compared with baseline. The reductions were significant compared with the placebo group ($p < 0.05$).No significant differences were observed between the two groups with regard to mean changes of the activity of plasma protein C, coagulation factors V, VII, IX, and serum PAI-1 to tPA ratio.

Conclusion: L-Carnitine supplement reduces serum CRP, a marker of systemic inflammation, and plasma fibrinogen, an inflammation-related coagulation factor, in hemodialysis patients. Therefore, L-carnitine may play an effective role in preventing cardiovascular diseases in these patients.

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NUTRITION-RELATED CARDIOVASCULAR DISEASE RISK FACTORS IN CHRONIC KIDNEY DISEASE: RELATIONSHIP WITH CLINICAL OUTCOME

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The significance of nutrition-related risk factors in early-stage chronic kidney disease (CKD) is largely unknown. Evidence in end-stage disease indicates a 'risk-factor paradox' with traditional cardiovascular (CV) risk factors associated with improved survival. This study aims to assess the relationship between nutrition-related risk factors and clinical outcome in early CKD.

All patients with eGFR 15-60 ml/min commencing care at a large metropolitan hospital in 2008 and 2009 were assessed for a number of traditional (body mass index, waist circumference, lipids LDL, HDL, triglycerides) and renal-specific (serum phosphate (PO_4), 25(OH)Vitamin D, albumin and haemoglobin) risk markers. Serum creatinine, eGFR, PTH, calcium, comorbidities, age and gender were also collected from medical records. Clinical outcome was defined as reaching renal-end points (death, dialysis commencement and/or doubling serum creatinine) by June 30, 2011. Univariate analysis was undertaken by t-test and multivariate survival analysis by Cox time-dependant hazards model.

667 patients were investigated. During follow-up (median=18; range 1-40 months) 36% (n=239) were discharged from care or lost to follow-up. Of the 428 patients remaining 25% (n=106) a renal- end point of death(13%), dialysis (7%) or doubling creatinine (5%). In a univariate analysis, PO_4 (event 1.35; 95%CI 1.29-1.40 vs. no event 1.15; 1.13-1.17 mmol/L), Vitamin D (61.7; 54.0-69.4 vs 75.8; 72.8-78.8 ng/mL) and serum albumin (34.2; 32.8-35.6 vs 38.7; 38.3-39.1 g/L) were related to outcome renal-end point. In the survival analysis model, only PO_4 (HR 8.9; 95%CI 3.3-24.5) and Albumin (0.90; 0.87 – 0.93) were independently associated with outcome, after adjusting for a range of confounding factors.

Traditional CV-risk factors in this CKD population were not associated with clinical outcome. Despite being within clinical reference range, serum phosphate and albumin were independently associated with clinical outcome. This may highlight a potential therapeutic target for risk management to delay or prevent renal end-points in CKD.

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EVALUATION OF THE URINARY SODIUM EXCRETION IN PATIENTS WITH LOW SODIUM DIET

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One of the factors involved in the progression of chronic kidney disease (CKD) is the intake of salt. The ratio of salt to hypertension, cardiovascular and cerebrovascular diseases has been extensively demonstrated in several studies. The purpose of this study was to estimate sodium intake in a group of patients with CKD and compared with the urinary excretion of sodium in patients with CKD of any cause at all stages of kidney disease. The design was a cross-sectional observational study, reflecting the initial moment of a protocol of a randomized, prospective and controlled study (Salted). On the same visit was also conducted to collect a food recall. The dietary sodium intake was calculated from the 3-day food record using the Software Avanutri®. For the analysis of sodium added to foods, each 1000mg of salt purchased for the family was divided by the number of people living with the patient, and the result was divided by the number of days that the patient reported the duration of salt until the next purchase. Urinary sodium was measured in urine samples from 24 hours through automated method (CI-8200 Architect - Abbott Diagnostics). After this analysis, we performed a correlation between food records provided by the patient and the result of urinary sodium excretion. Forty-one patients were included, with glomerular filtration rate averaged 38.83 $\pm$ 13.63 ml / min. The data correlation between the questionnaire data to assess the sodium intake (food record) show a