5 and 23% used 10 guidelines. Only one dietitian applied all guidelines. A total compliance to guidelines score was developed as a percentage of total compliance which showed 37 ± 15.51% (min14, max 73). Barriers identified were lack of time and lack of integration into the medical team. It is evident that dietetic practices in Lebanon targeting HD patients need support in all aspects: time, knowledge, empowerment and training.

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ROUTINE HOSPITAL SCREENING TOOL (NRS-2002) IS ADEQUATE FOR IDENTIFYING MALNOURISHED DIALYSIS PATIENTS IN MALAYSIA.

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Malnutrition is prevalent (> 50%) in maintenance hemodialysis (HD) and continuous ambulatory peritoneal dialysis (CAPD) patients in Malaysia. The priority is to identify a suitable nutritional screening tool for use in dialysis units. We conducted a study between June to November 2011 testing simplified nutritional screening tools on 155 HD and 90 CAPD patients to validate their potential application. Routine nutritional screening tools commonly used for hospitalized patients (NRS–2002, MST, MUST) were compared against tools specific to dialysis such as the malnutrition-inflammation score (MIS), modified Subjective Global Assessment (mSGA) and geriatric nutritional risk index (GNRI) with diagnostic values inclusive of anthropometry, biochemistry and dietary itnake. The mSGA compared to MIS indicated greater specificity for BMI < 25 kg/m^2, serum albumin < 40 g/dL, AMA ≤ 15th percentile, energy intake < 30 kcal/kg/day and protein intake < 1.2 g/kg/day with greater sensitivity scoring (99.3 *vs* 80.4%, 98.6 *vs* 72.9%, 99.3 *vs* 72.9%, 98.2 *vs* 67.3%, 98.3 *vs* 68.5% respectively). Comparing hospital screening tools against mSGA or MIS as reference standards for both PD and CAPD patients, the receiver operating characteristic (ROC) curve analysis indicated greater sensitivity in predicting malnutrition was associated with NRS (averaging at 58% against mSGA and 69% against MIS) whilst all other instruments were less predictive. GNRI, developed for Japanese dialysis patients was least sensitive to detect malnutrition. In conclusion, NRS–2002 can be utilized for identifying malnourished dialysis patients in Malaysia.

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A NEW NUTRITIONAL SCREENIG TOOL; GNRI IN JAPANESE INCIDENT DIALYSIS PATIENTS.

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Background: Inflammation and malnutrition are common in chronic kidney disease (CKD) patients and among strong predictor of cardiovascular disease (CVD). Geriatric nutritional risk index [GNRI = 1.489 × albumin (g/dl) + 41.7(body weight (BW) / ideal BW)] has been reported to be a simple and accurate nutritional screening tool in CKD patients as well as old people. Previous studies demonstrated low lymphocyte count (LLC) predicts poor outcome in patients with CVD and heart failure.

Methods: A total of 68 Japanese incident dialysis patients (44 males, mean age 61 ± 11 years) were enrolled and followed in an ongoing prospective study. Nutritional status [albumin, body mass index (BMI), abdominal circumstances, subjective global assessment (SGA) and GNRI] was assessed. Inflammatory biomarkers including white blood cell count (WBC) and differential were determined.

Results: The median of GNRI was 95.4. When divided into two groups by the median, there were no association in age, gender, diabetes, SGA, medications including ESA between two groups. GNRI showed significantly correlation to hemoglobin ($\rho = 0.31$, $p = 0.014$) and did negatively correlation to WBC count ($\rho = -0.24$, $p = 0.049$) and ferritin ($\rho = -0.27$, $p = 0.021$) in Spearman's rank test. Although these associations disappeared 1 year after initiation of dialysis therapy, the patients with higher incremental GNRI significantly showed increased lymphocyte count.

Conclusion; Our results suggest that GNRI is a simple nutritional tool for dialysis patients and that GNRI might detect inflammation and predict CVD.

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MUSCLE ATROPHY IN PATIENTS WIRH CKD RESULTS FROM FGF23/ KLOTHO-MEDIATED SUPRESSION OF INSULIN/IGF-I SIGNALING

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Muscle atrophy is a significant consequence of chronic kidney disease (CKD) that increases a patient's risk of mortality and decrease their quality of life. In CKD patients, the circulation levels of FGF23 are significantly increased, but the exact pathological significance of the increase and relationship between FGF23 and muscle atrophy are not clear. Because of Klohto, acts as a co-receptor of FGF23 is detectable in limited tissues including in kidney and brain, but not in skeletal muscles. In contrast, recently reports indicated that the extracellular domain of klohto is cleavage for some reason on the cell surface and detected in the blood in animals. In this study, we attempted to identify the causative factors responsible for the shedding of Klotho, and whether both FGF23 and Klohto induced muscle atrophy via reduction of insulin/IGF-I signaling. We first investigated by treating kidney cells with various factors related in pathological factors in CKD. As a result, we found that advanced glycation endproducts (AGEs), an accumulated in patients with CKD and diabetes mellitus, increases shedding of Klohto in kidney cells. It is common knowledge that insulin/IGF-I signaling is necessary for normal skeletal growth. As a result, we showed that both FGF23 and Klohto inhibited differentiation of cultured skeletal muscle cells through down-regulation of insulin/IGF-I signaling. These observations suggested a divergent role of FGF23 and soluble klohto in the regulation of skeletal muscle differentiation and thereby muscle atrophy under pathological conditioned in CKD patients. Our results further imply that FGF23/Klohto may serve a new therapeutic target for CKD-induced muscle atrophy.

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EFFECTS OF DIETARY SALT RESTRICTION ON PUROMYCIN AMINONUCLEOSIDE NEPHROSIS

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Dietary salt restriction may reduce proteinuria although the mechanism is not clear. We investigated the effects of dietary salt restriction on rat kidneys in an animal model of glomerular proteinuria. Male Sprague-Dawley rats were divided into 3 groups: vehicle-treated normal-salt controls, puromycin aminonucleoside (PA)-treated normal-salt rats, and PA-treated low-salt rats. PA was given at a dose of 150 mg/kg BW at time 0, followed by 50 mg/kg BW on days 28, 35, and 42. Sodium-deficient rodent diet with and without additional NaCl (0.5%) were provided for normal-salt rats and low-salt rats, respectively. On day 63, kidneys were harvested for histopathologic examination. PA treatment produced overt proteinuria and renal damage. Dietary salt restriction insignificantly reduced proteinuria in PA-treated rats, and PA-treated low-salt rats had lower urine output and smaller creatinine clearance than vehicle-treated normal-salt controls. The tubulointerstitial injury score positively correlated with proteinuria. It was significantly increased by PA treatment and relieved by low-salt diet. ED1-positive infiltrating cells and immunostaining for interstitial collagen III were significantly increased by PA treatment. These changes appeared less in PA-treated low-salt rats although the differences in PA-treated normal-salt versus low-salt rats did not reach the statistical significance. Our results suggest that renal histopathology in PA nephrosis may potentially be improved by dietary salt restriction. The decreasing tendency in glomerular filtration rate induced by low-sodium diet might be beneficial in retarding renal progression.

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