

low serum albumin, high phosphorous, high C-reactive protein level, high mean arterial pressure, low ejection fraction and high left ventricular filling pressure (E/E') were also frequent in higher cTnT group in simple analysis. Using cut point of cTnT level ≥ 0.1 ng/ml, which was known as a valuable predictor of all-cause and CV mortality in many studies, multiple logistic regression analysis was undertaken for adjusting interaction among independent variables. After adjusting confounding factors, $\log_{25}(\text{OH})\text{D}$ was independently associated with elevated cTnT level (OR, 0.148; 95% CI, 0.026 to 0.832). Male sex and presence of DM were also significant independent variables (OR, 5.025; 95% CI, 1.566 to 16.125 and OR, 4.278; 95% CI, 1.315 to 13.915, respectively). However, other variables, including $\log_{1.25}(\text{OH})_2\text{D}$ did not show significance in this regression model. In conclusion, low 25(OH)D level was associated with elevated cTnT level even in Korean ESRD patients, suggesting 25(OH)D deficiency partly plays a role in poor CV outcome in this population like Western population.

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192 THE ASSOCIATION OF GERIATRIC NUTRITIONAL RISK INDEX AND TOTAL LYMPHOCYTES COUNT WITH MORTALITY IN KOREAN HEMODIALYSIS PATIENTS

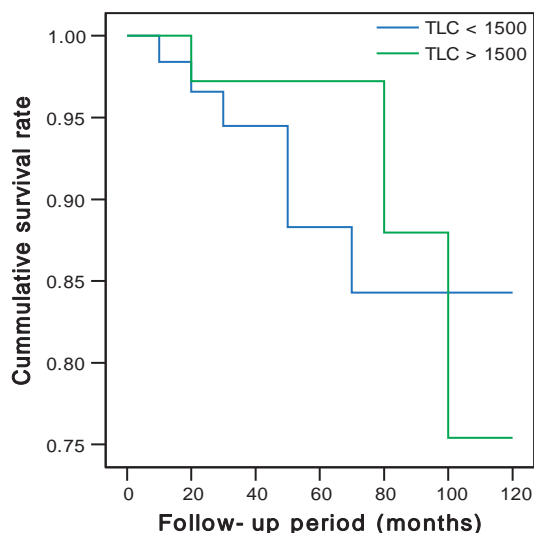
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Background: Our objective was to examine the association between the Geriatric Nutritional Risk Index (GNRI) and Total lymphocytes count (TLC) with mortality in Korean Hemodialysis Patients. **Methods:** We examined the GNRI and TLC of 120 maintenance hemodialysis patients and followed these patients for 120 months. Predictors for all-cause death were examined using life table analysis and the Cox proportional hazards model. **Results:** Life table analysis revealed that subjects with a GNRI < 90 ($n = 19$) had a lower survival rate than did those with a GNRI ≥ 90 ($n = 101$) (Wilcoxon test, $P = 0.048$), but subjects with a TLC $< 1500/\text{mm}^3$ ($n = 76$) had a similar survival rate compared those with a TLC $\geq 1500/\text{mm}^3$ ($n = 44$) (Wilcoxon test, $P = 0.500$). Multivariate Cox proportional hazards analyses demonstrated that the GNRI was a significant predictor of mortality [hazard ratio (HR) 9.315, 95% confidence interval (CI) 1.161–74.753, $P = 0.036$], after adjusting for age, sex, presence of type 2 diabetes mellitus, Kt/V, nPCR and TLC. The association of a GNRI ≥ 90 with a TLC $\geq 1500/\text{mm}^3$ seemed to exclude the occurring of complications with moderate reliability.

Conclusion: These results demonstrate that the GNRI may be a significant predictor of mortality in Korean hemodialysis patients. However, the use of TLC might improve the evaluation of nutritional risk and the identification of patients at risk of malnutrition. Figure 1. Total lymphocytes count and 120-month survival of hemodialysis patients. In both groups, survival rate during the follow-up period was similar. (life table analysis, $P = 0.500$).



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193 NATURAL CORRECTION OF HYPERURICEMIA, NOT BY ALLOPURINOL, COULD SLOW DOWN THE PROGRESSION OF RENAL DISEASE IN THE PATIENT WITH CHRONIC KIDNEY DISEASE STAGE 3

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Purpose: Correction of hyperuricemia can slow down the progression of renal disease in animal study. However, there is limited data regarding the effect of lowering serum uric acid in patients with hyperuricemic chronic kidney disease (CKD) stage 3 (estimated GFR [eGFR] 30–60 mL/min).

Methods: We retrospectively investigated 100 patients (age 56.1 ± 9.9 years, M:F = 79:21) with hyperuricemia (serum uric acid 7.9 ± 0.9 mg/dL) and CKD stage 3 (eGFR 52.0 ± 7.0 mL/min) 10 years ago. First, to evaluate the effect of allopurinol on the progression of renal disease, 23 patients who have taken allopurinol were compared with the randomly selected 23 patients according to the level of serum uric acid and eGFR as a control group among the other 77 patients who have never received allopurinol. Second, to clarify the effect of lowering serum uric acid by diet on renal disease progression, these 77 patients were divided into 2 groups by decreased amount of serum uric acid during 10 years and compared each other (group 1 > 1.0 mg/dL vs. group 2 < 1.0 mg/dL).

Results: First, although serum uric acid levels were significantly decreased (8.5 ± 1.0 to 6.6 ± 1.2 mg/dL, $p < 0.001$) in subjects treated with allopurinol, the change of eGFR was not significant (48.8 ± 8.6 to 44.5 ± 18.5 mL/min, $p = 0.230$). In control group, serum uric acid was also decreased (8.2 ± 1.0 to 7.3 ± 1.6 mg/dL, $p = 0.025$) but eGFR did not change significantly (49.0 ± 8.7 to 50.3 ± 20.2 mL/min, $p = 0.726$). Second, eGFR increased significantly (54.0 ± 6.0 to 63.1 ± 18.0 mL/min, $p = 0.002$) in the group 1 whereas eGFR decreased (51.9 ± 6.4 to 46.1 ± 15.6 mL/min, $p = 0.008$) in the group 2 after 10 years. In the multivariate linear regression analysis in the entire cohort ($n = 100$), age ($p = 0.023$), change of serum creatinine ($p = 0.001$), and change of serum uric acid ($p < 0.001$) were found to be significant factors that influence the change of eGFR during study period.

Conclusions: We identified the protective effect of lowering serum uric acid against progression of renal disease in patients with CKD stage 3. However, allopurinol itself did not have such beneficial effect.

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194 WHAT TO EAT. THE TERAPEUTIC AND DIETETIC COMPLIANCE OF PATIENTS ON PERITONEAL DIALYSIS.

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Nutrition education is important for patients on peritoneal dialysis. Yet, despite initial nutrition training and monthly reinforcements during follow-up visits, phosphorus control remains unsatisfactory. For this reason a meeting with an external renal dietician open to patients and their relatives was organized. Biographical data and average phosphorus levels for the 3 months prior to the meeting are shown in Table.1

Patients	11 (6 F - 5 M)
Age (years)	63 (47-81)
Time on Dialysis (months)	72 (6-348)
Body Mass Index (kg/m ²)	25.8 (22.3-32.4)
Phosphorous mg/dl (average of 3 months)	5.95 (3.9-8.2)

After a lecture delivered by the centre's nephrologist, a questionnaire on phosphorus control was compiled by the participants. The renal dietician then explained how to: control dietary phosphorus intake, properly use chelating agents, reduce phosphorous in cooking and make smart food choices. A trained chef then demonstrated how to cook some regional recipes specifically reworked to reduce their phosphorous content without sacrificing taste. Participants then verified that the recipes were indeed appetizing. At this point, the same questionnaire given at the start of the meeting was re-given. From this, an improvement in the understanding of phosphorus control was seen. For example, in response to the question "Who is responsible for controlling phosphorus?" the percentage of patients correctly answering "the patient" rose from 55% before the meeting to 91% after the meeting. To successfully manage kidney disease