RESULTS: Patients (n=76) with LVH as compared to patients with no LVH (n=42) were older in age (66±14 vs. 55±16 years; p<0.001), had lower sMg (2.1±0.3 vs. 2.4±0.4 mg/dl; p<0.001) and higher sCa (9.4±0.6 vs. 9.1±0.7 mg/dl; p<0.05), malnutrition-inflammation index (5.0±2.8 vs. 4.0±2.0; p<0.05), body mass index (27.4±5.1 vs. 24.1±3.4; p<0.001), prevalence of diabetes (81.2 vs.18.8; p<0.05), coronary artery disease (79.4 vs.20.6; p<0.05) and peripheral vascular disease (78.3 vs. 21.7%; p<0.01). In a multivariate logistic regression analysis adjusted for all factors mentioned above, each increase of sMg by 1 mg/dl was associated with 89% (OR= 0.11, 95% CI: 0.03-0.41; p<0.001) lower odds of having LVH. sMg was inversely associated with LVMI (r=-0.21; p<0.05) and in a forward stepwise multivariate model (R²= 0.209; p<0.001), sMg emerged as a strong independent predictor of LVMI (p<0.01) explaining about 4% of its variance. The area under the ROC curve for predicting the development of LVH was 0.697 (p<0.001) and at an optimal sMg cutoff of 2.22 mg/dl the sensitivity and specificity of sMg in predicting the occurrence of LVH were 66 % and 69 %, respectively. Considering LV geometry, there was a progressive decrease in sMg from the normal group (2.39±0.4 mg/dl) to concentric remodeling (2.37±0.41 mg/dl), eccentric (2.19±0.31 mg/dl) and then to concentric (2.08±0.36 mg/dl) group (p<0.01 for the trend).

CONCLUSIONS: A lower sMg is a major determinant of echocardiographic LVH. Prospective studies may determine whether therapeutic adjustments of sMg can prevent or reduce the risk of LVH in stage 5 CKD patients.