assigned as the baseline level. Serum creatinine was measured again within 48 hours after measurement of urine [TIMP-2]•[IGFBP-7]. The outcome was acute kidney injury based on the KDIGO criteria.

Results: The study included 86 patients, 55.8% male, with a mean age of 54 years old. In terms of COVID severity, 50% were moderate, 27.9% were severe, while 22.1% were critical. Among the 86 participants in the study, 79 (91.9%) did not develop acute kidney injury. Among the 7 (8.1%) participants who developed AKI, four had stage 1, one had stage 2, and two had stage 3. There was a high percentage of mortality at 71.4% among patients who had AKI, while majority or 86.1% of patients without AKI were discharged from the hospital. The cutoff value of [TIMP-2]•[IGFBP-7] to predict AKI was 0.59 ng/mL^2/1000 and the AUC was 0.62 (95%, confidence interval 0.38-0.87). The sensitivity of this cut-off was 57.1% (95%, confidence interval 56.0-58.3) with a specificity of 74.7% (95%, confidence interval 74.4-75.0). A separate analysis was done to determine the ability of [TIMP-2]•[IGFBP-7] to predict moderate to severe AKI. This analysis yielded an AUC of 0.78 (95%, confidence interval 0.51-1.0) with an optimal cut-off value of 0.29 for screening for AKI.
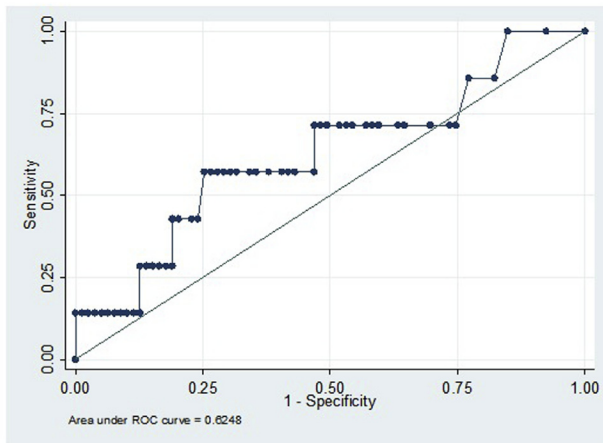

Figure 1. Urinary [TIMP-2]·[IGFBP-7] test area under the receiver operating characteristic curve (AUC) of 0.62 for discriminating COVID-19 patients with acute kidney injury (AKI) (n = 7) from those with no AKI (n = 79)
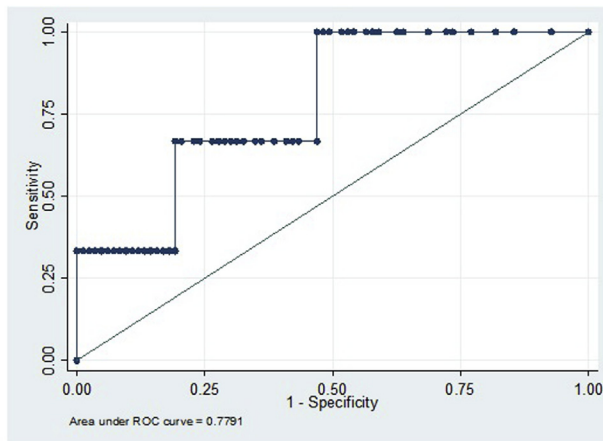

Figure 2. Urinary [TIMP-2]·[IGFBP-7] test area under the receiver operating characteristic (ROC) curve (AUC) of 0.78 for discriminating COVID-19 patients with moderate to severe acute kidney injury (AKI) (n = 3) from those with stage 1 AKI or no AKI (n = 83)

Conclusions: Urine [TIMP-2]•[IGFBP-7] is moderately accurate in predicting stage 2 to 3 acute kidney injury among patients hospitalized with moderate, severe or critical COVID-19, but larger studies with a larger percentage of patients developing AKI are recommended to validate these findings.

No conflict of interest

POS-076

SERUM CYSTATIN C AS EARLY MARKER FOR AKI OF PATIENTS AFTER CORONARY ANGIOGRAPHY: A PROSPECTIVE, OBSERVATIONAL STUDY IN MEXICAN POPULATION

Rizo Topete MD, L*[1,2], Alvarado-Villarreal, F[3], José Ramón, A[4], Urrutia-Stamatio, B[4], Villegas-Mejía San Pedro Garza Garcia, M[3]

[1]UANL- Hospital Universitario "Jose Eleuterio Gonzalez", Nefrologia, Monterrey, Mexico; [2]UDEM- Hospital Christus Muguerza Alta Especialidad, Internal Medicine and Critical Care, Monterrey, Mexico; [3]UDEM- Hospital Christus Muguerza Alta Especialidad, Internal Medicine and Critical Care, San Pedro Garza Garcia, Mexico, [4]UDEM- Hospital Christus Muguerza Alta Especialidad, Cardiology, San Pedro Garza Garcia, Mexico

Introduction: Cystatin C is only filtered and metabolized in the kidney, which makes it the ideal marker of kidney function. High sensitivity and specificity for early detection of acute kidney injury and contrast induced kidney injury (C-AKI). Early identification of C-AKI after percutaneous coronary intervention may help to change the prognosis of this patients.

Methods: Prospective, observational study, 21 patients, Feb-2020 to Jan-2021. Capillary serum samples prior to contrast administration. Cystatin C was quantified by quantitative immunofluorescence analysis (GETEIN1100). Acute kidney injury (AKI) was defined by the KDIGO-AKI criteria. Follow up with creatinine every 24 hours for up to 72 hours. Primary outcome was to determine the incidence of acute kidney injury, secondary outcome was to determine comorbidities and epidemiological risk factors.

Results: From the 21 patients, 14 men (67%), 7 women (33%), the average age was 63 years. 28.5% had diabetes mellitus, 74.5% hypertension, 38.1% ischemic heart disease, and 4.6% had a history of cancer. Glomerular filtration rate by MDRD was 76.27 ml/min in the group without AKI and 68.7 ml / min in the group with AKI, without statistically significant differences (p= 0.56). There was statistically significant difference between both groups in the serum tests: Cystatin C 1.11 vs 1.20 (p= 0.047), Creatinine at 48h 0.91 vs 1.64 (p= <0.001), Phosphorus 3.81 vs 4.8 (p= 0.024), Glucose 105.86 vs 134.6 (p= 0.021), Cholesterol 178.7 vs 117.7 (p= 0.046), Iron 77.8 vs 45.7 (p= 0.027). It was divided into interquartile groups of cystatin C; Q1 (<0.93 mg / dl), Q2 (0.93-1.16 mg / dl), Q3 (1.17-1.69 mg / dl), Q4 (> 1.69 mg / dl) where the incidence is 23.5%, 50%, in Q3 we have no data, and 50% respectively.

Conclusions: We founded higher incidence of C-AKI than previously reported. Major risk factors were hyperglycemia, iron deficiency and low cholesterol levels. Cystatin C use must continue to grow, it can be efficiently used as a screening tool in the Cath lab.

No conflict of interest

POS-077

CD153-CD30 SIGNALING ACCELERATES AGE-DEPENDENT TERTIARY LYMPHOID TISSUE FORMATION AND KIDNEY INJURY

Sato, Y*[1,2], Oguchi, A[1], Toriu, N[1], Taniguchi, K[1], Yoshikawa, T[1], Komidori, S[1], Hosoi, T[2], Shimizu, Y[1], Kondo, M[1], Boor, P[3,4], Floege, J[4], Yanagita, M[1,5]

[1]Graduate School of Medicine- Kyoto University, Department of Nephrology, Kyoto, Japan; [2]Graduate School of Medicine- Kyoto University, Medical Innovation Center TMK Project, Kyoto, Japan; [3]RWTH University of Aachen, Institute of Pathology, Aachen, Germany; [4]RWTH University of Aachen, Department of Nephrology, Aachen, Germany, [5]Graduate School of Medicine- Kyoto University, Institute for the Advanced Study of Human Biology ASHBi, Kyoto, Japan

Introduction: The elderly show a reduced capacity for renal regeneration after acute kidney injury (AKI). We previously showed that, after AKI, aged, but not young, kidneys exhibit tertiary lymphoid tissues (TLTs), which underlie maladaptive repair in aged injured kidneys. TLT is an unique microenvironment for lymphocyte activation and differentiation *in situ* and are involved in pathophysiology in various human diseases. However, the cells and signals responsible for age-dependent TLT formation in the kidneys are still undefined.

Methods: We investigated immune cells in aged injured kidneys with TLTs, 45 days after ischemic reperfusion injury, utilizing scRNAseq, bulk RNAseq and repertoire analysis, combined with flow cytometry