AKIs were more related to immunologic response and had significantly higher mortality. Patients who died had significantly higher ferritin and d-dimer levels upon their hospital admissions (p=0,000). Electrolyte disturbances, metabolic acidosis and mortality were also higher in patients who developed AKI later. Hypernatremia (OR: 6,5, 95% CI: 3 – 13,9) and phosphorus disturbances (both hyperphosphatemia (OR: 3,3; 95%CI: 1,6 – 6,9) and hypophosphatemia (OR: 3,9; 95% CI: 2,0-7,9)) were related to mortality.
CONCLUSION: Findings of this study suggest that AKI in COVID-19 is not of one kind. When developed, AKI should be evaluated in conjunction with the disease stage and possible etiologies

MO371 **TIME AND THE ETIOLOGY OF ACUTE KIDNEY INJURY DEFINE PROGNOSIS IN THE COURSE OF COVID-19**

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BACKGROUND AND AIMS: Kidneys are among the affected organs in COVID-19 and there may be different etiologies resulting in acute kidney injury (AKI) in different stages of the disease. There have been previous studies focusing on incidence and mortality of AKI in COVID-19 but none has made in depth analysis in relation to the background pathophysiology. Based on previous observations, we hypothesized that all AKIs seen in COVID-19 are not uniform and we aimed to analyze the etiologies and prognosis of AKI among hospitalized COVID-19 patients in relation to the time of AKI during different phases of the disease.
METHOD: A total of 1056 patients were admitted to the designated COVID-19 clinics from March to July in 2020. 77 Patients who were younger than 18 years old and 7 kidney transplant patients were excluded from the study. 427 of the remaining patients were confirmed by real time polymerase chain reaction (RT-PCR) test.). As eGFR below 60 mL/min/1,73 m^2 was already shown to be related to mortality, these patients (44) were also excluded. As immunologic response is generally accepted to start with the second week of COVID-19 course, patients were classified into three groups, those who had AKI on admission, those who developed AKI in the first week and those who developed AKI starting from 7^{th} day. Initial lymphocyte counts, creatinine levels, electrolytes, acid-base status and changes in the inflammatory markers were compared between the groups. A comparison between patients who survived and who died was also performed.
RESULTS: 89 of the 383 included COVID-19 patients developed AKI. 24% of those who developed AKI died. Patients who developed AKI later had higher peak CRP and D-dimer levels with lower nadir lymphocyte counts (p=0,000, 0,004 and 0,003 respectively). Additionally, patients who died had higher initial inflammatory marker levels and lower lymphocyte counts than those who survived. Mortality of patients who had AKI on hospital admission (13%) was similar to the overall COVID-19 mortality for inpatients, however it was as high as 44% for those who developed AKI after 7^{th} day. Early AKI was related to pre-renal causes and had a milder course. However, later