

Assess the Association between AKI and the Incidence of Remission and Relapse of Proteinuria

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Description

One of the most prevalent primary nephrotic syndromes is called Minimal Change Disease (MCD). Acute Kidney Injury (AKI) is more common in MCD than in membranous nephropathy, and previous studies demonstrated that kidney survival was significantly better in MCD than in focal segmental glomerulosclerosis and membranous nephropathy. A clinical effect of AKI in MCD is still unknown. Conflicting findings regarding an association between AKI and the incidence of remission of proteinuria in 43, 53, and 78 MCD patients in Taiwan, Japan, and the United Kingdom, respectively, were found in several retrospective cohort studies. The UK study found no link between AKI and the occurrence of proteinuria relapse. The Japan Nephro we assumed that the serum creatinine level before the onset of MCD was at the same serum creatinine level 2 months after initiating IST, which was used to estimate the estimated Glomerular Filtration Rate (eGFR) before the onset of MCD. This assumption was based on the fact that 108 (95.6%) and 96 (85.0%) patients achieved non-nephrotic proteinuria of urinary protein.

Clinical Effect of AKI on Relapse of Proteinuria after Remission

According to the Improving Global Outcomes Clinical Practice Guideline for AKI7, a baseline AKI is one in which the serum creatinine level has increased by at least 0.3 mg/dl, or 50%, from the serum creatinine level prior to presentation. The stages of AKI were as follows: The first stage is a rise in serum creatinine of at least 0.3 mg/dl and/or 50 to 99 percent; Stage 2 is a 100% to 199% rise in serum creatinine levels; and stage 3 is a serum creatinine level that has gone above or equal to 4.0 mg/dl or has gone up by at least 200 percent. The clinical effect of AKI on relapse of proteinuria after remission, which was defined as urinary protein of at least 1.0 g/day and/or dipstick urinary protein 2+ continued 2 or more times, in typical patients with MCD who achieved remission within 2 months of IST, is also assessed. The baseline characteristics of 113 patients with MCD stratified by baseline AKI stage. Twenty (17.7%), eleven (9.7%), and six (5.3%) patients with AKI stages 1, 2, and 3 were among

the 37 patients with baseline AKI. Body mass index, systolic blood pressure, serum creatinine level, eGFR, and urinary protein level showed significant differences between the four groups at the same baseline AKI stage. Within one month of IST, 111 patients (98.2%), 31 patients (27.4%), 15 patients (13.3%), and 2 patients (1.8%) received oral prednisolone, intravenous methylprednisolone, cyclosporin, and rituximab, respectively. The pre-presentation eGFR and serum creatinine level were not significantly different between the four groups of AKI stage at baseline. Two patients in the group with no AKI developed end-stage kidney disease 1.2 and 2.0 years after beginning IST. Prepresentation eGFR was not significantly associated with the incidence of remission whereas patients with a higher baseline AKI stage were more likely to have a lower cumulative probability of remission. Patients with baseline AKI stage 2 had a significantly lower cumulative probability of remission than patients without AKI, according to an unadjusted model. Prepresentation eGFR was not found to be a predictor of late remission after the multivariable adjustment, but baseline AKI stages 2 and 3 were found to be predictors of late remission along with age and serum albumin level. Adult patients with MCD have been the subject of a number of small retrospective cohort studies examining the relationship between AKI and proteinuria remission. A Japanese retrospective cohort study evaluated the association between AKI and the incidence of remission, after adjusting for clinically relevant factors. This study elaborately defined AKI as an increase in serum creatinine level to at least 1.5 times the baseline level known or presumed to have occurred within the prior 7 days during the 4 weeks after initiating IST.

Clinical Effect of AKI on MCD

After adjusting for clinically relevant factors, this study further defined AKI as an increase in serum creatinine level to at least 1.5 times the baseline level known or presumed to have. According to the Kidney Disease, patients were divided into three groups: no-AKI, AKI stage 1 or 2, and AKI stage 3: This study clarified a dose-dependent association between AKI stage and the incidence of remission in multivariable-adjusted Cox proportional hazards models, which improved Global Outcomes classification. When compared to patients without AKI, even

those with AKI stage 1 or 2 had a significantly lower cumulative probability of remission. Additionally, we were able to identify AKI stage 2, not stage 1, as a significant suppressor of remission due to the study's larger sample size. The incorrect classification of patients with baseline AKI and no improvement in eGFR two months after initiating IST into the no-AKI group was one of the study's potential biases. This misclassification undervalued the clinical effect of AKI on remission of proteinuria because AKI delayed MCD remission, suppressing remission in the no-AKI

group and promoting remission in the AKI group. It's possible that the actual impact of AKI is greater than what the current study suggests. In conclusion, adult MCD patients with AKI stage 2 or higher were significantly less likely to enter remission, according to the Japan Nephrotic Syndrome Cohort Study. However, the clinical effect of AKI on MCD should be confirmed in large, well-designed studies due to the observational nature of the Japan Nephrotic Syndrome Cohort Study.