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The potential role of IL-33/ST2 in renal allograft rejection

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Objectives: Interleukin (IL)-33 and its receptor, growth stimulation gene-2 (ST2) are increased in allograft rejection. Recent studies showed that upregulation of IL-33 has protection effect against chronic allograft rejection. We hypothesized that IL-33 and ST2 expression differently express according to rejection type, acute antibody mediated rejection (AAMR), acute cellular mediated rejection (ACMR), and chronic antibody mediated rejection (CAMR).

Methods: Kidney biopsy tissue were obtained from healthy controls (n=4) and kidney transplanted recipients with ABMR (n=10), ACMR (n=10), and CAMR (n=10). We evaluated the tissue expression of IL-33 and ST2 in allograft biopsies from 4 groups by quantitative immunostaining.

Results: The expressions of ST2 were significantly increased in AAMR (p=0.014), ACMR (p=0.028), and CAMR (p=0.036), compared to healthy controls. On the contrary, IL-33 expressions attenuated in kidney tissue with AAMR (p=0.028) and CAMR (p=0.014). IL-33 increased in ACMR group but did not show statistical significance (p=0.580). Interestingly, IL-33 and ST2 had a positive correlation in AAMR (r=0.692, p<0.001) and ACMR (r=0.661, p<0.001), and a negative correlation in CAMR (r=-0.459, P=0.011).
IL33 and ST2 levels increased after transient hypoxia via hypoxia-inducible factor (HIF)-1α in primary cultured human renal tubular cells.

Conclusions: The up-expression of ST2 can be a stable hallmark of acute and chronic rejection in kidney recipient. IL-33 expression in the allograft tissue with rejection may be increased during early phases of rejection, as an alarm marker. Furthermore, we need to evaluate the role of the IL-33/ST2 axis in allograft rejection.