CD55/Crry and complement C3d expression during microvascular rejection in mouse airway allografts

Mohammad Afzal Khan, Talal Shamma, Hala Abdulrahman Ahmed, Abdallah Mohamed Assiri and Dieter Clemens Broering
King Faisal Specialist Hospital and Research Centre, Riyadh, Saudi Arabia

Complementary regulatory proteins (CRPs), including CD55 and complement receptor 1-related gene/protein y (Crry) primarily regulate toxic effects of complement activation. During cognate T cell/antigen presenting cell interactions, active complement fragments are up-regulated, and CRPs are down-regulated. Here, we demonstrated the change in vascular endothelial expression in CD55/Crry and C3d during microvascular loss in mouse airway allografts. BALB/c →C57BL/6 allografts were serially monitored for CD55, Crry and C3d deposition on CD31 positive vascular endothelial cells at d6, d8 and d10 post transplantation. In addition, all corresponding allografts were examined for tissue oxygenation, microvascular blood flow and functional microvasculature between donor and recipients during allograft rejection at same day points. We demonstrated that both CD55 and Crry down regulated during microvascular loss, while expression of C3d increased at d6–d10 post transplantation. Our results also demonstrated that subsequent increase in C3d on vascular endothelial cells was directly associated with microvascular associated injuries, graft hypoxia, ischemia during rejection. Together, these data indicate that stoichiometry of CD55/Crry and C3d expression has potential to affect microvascular associated injuries during the phase of rejection. These findings may be useful in designing anti-C3 therapy in combination with existing immunosuppressive regimens to rescue tissue/organ rejection.

mkhan26@kfshrc.edu.sa