The Association of Antibodies Against Angiotensin II Type 1 Receptor with Delayed Allograft Function of Kidney Transplantation

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Background: Delayed graft function (DGF) is a failure of the transplanted kidney to function immediately after transplantation mainly with causes of ischemia-reperfusion and immunological injury. The kidney graft from donor with high level of angiotensin II (Ang II) measured during procurement is significantly more likely to develop DGF. Ang II type I receptor (AT_1R) mediates most physiologic and pathophysiologic actions of Ang II, autoantibodies to AT_1R (anti-AT_1R) are implicated in several vascular pathologies. The impact of anti-AT_1R on clinic outcomes of DGF grafts was evaluated in this study.

Methods: We reviewed the records of all consecutive adult recipients who received single kidney transplantation and clinically management between Jan.2006 and Dec.2009 in our centre. The serum binding levels of anti-AT_1R were measured by a **r**ecombinant-receptor-based sandwich ELISA and a cutoff of 15 units was used to distinguish high from low binding.

Results: Three hundred and seventy-seven recipients were enrolled. The overall presence of DGF was 31%, and 12% recipients had high binding anti-AT_1R, the incidence of DGF in patients with high binding anti-AT_1R was significant higher than patients with low binding of anti-AT_1R (42% vs 29%, p=0.03). In addition, more female recipients, longer duration of renal replacement therapy, higher resistance index (RI) of allograft and more severe acute tubular injury/acute tubular necrosis were observed in recipients with DGF and high binding of anti-AT_1R (AT+DGF) comparing with recipients occurring DGF and low binding of anti-AT_1R (AT-DGF). The blood pressure and RI of allograft in AT+DGF recipients were ameliorated under the administration of angiotensin-converting enzyme inhibitors (ACEi) or Ang II blockers (ARB), and one-year graft survival and patient survival was similar between two groups.

Conclusions: Presence of high binding anti-AT_1 R had detrimental impacts on initiation and development of DGF.

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