25th Annual Scientific Meeting
Malaysian Society of Transplantation

TRANSPLANTATION: TRANSFORMING THE LANDSCAPE TOWARDS A NEW HORIZON

IMPACT OF DELAYED GRAFT FUNCTION AND OUTCOME OF CADAVERIC KIDNEY TRANSPLANT: A SINGLE-CENTRE EXPERIENCE.

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Introduction

Delayed graft function (DGF) is a predictor of poor long-term graft survival[1], but whether its effects are independent of rejection or higher serum creatinine (Scr) is controversial. This study aims to evaluate the impact of delayed graft function towards clinical outcomes post deceased donor kidney transplant (DDKT).

Method

The data of all patients who underwent DDKT in Hospital Selayang during a 3-year period (1st January 2019 till 31st December 2021), were evaluated retrospectively. In this study, delayed graft function is defined as the need of dialysis during the first week after transplantation. Descriptive results were analysed with SPSS version 27.

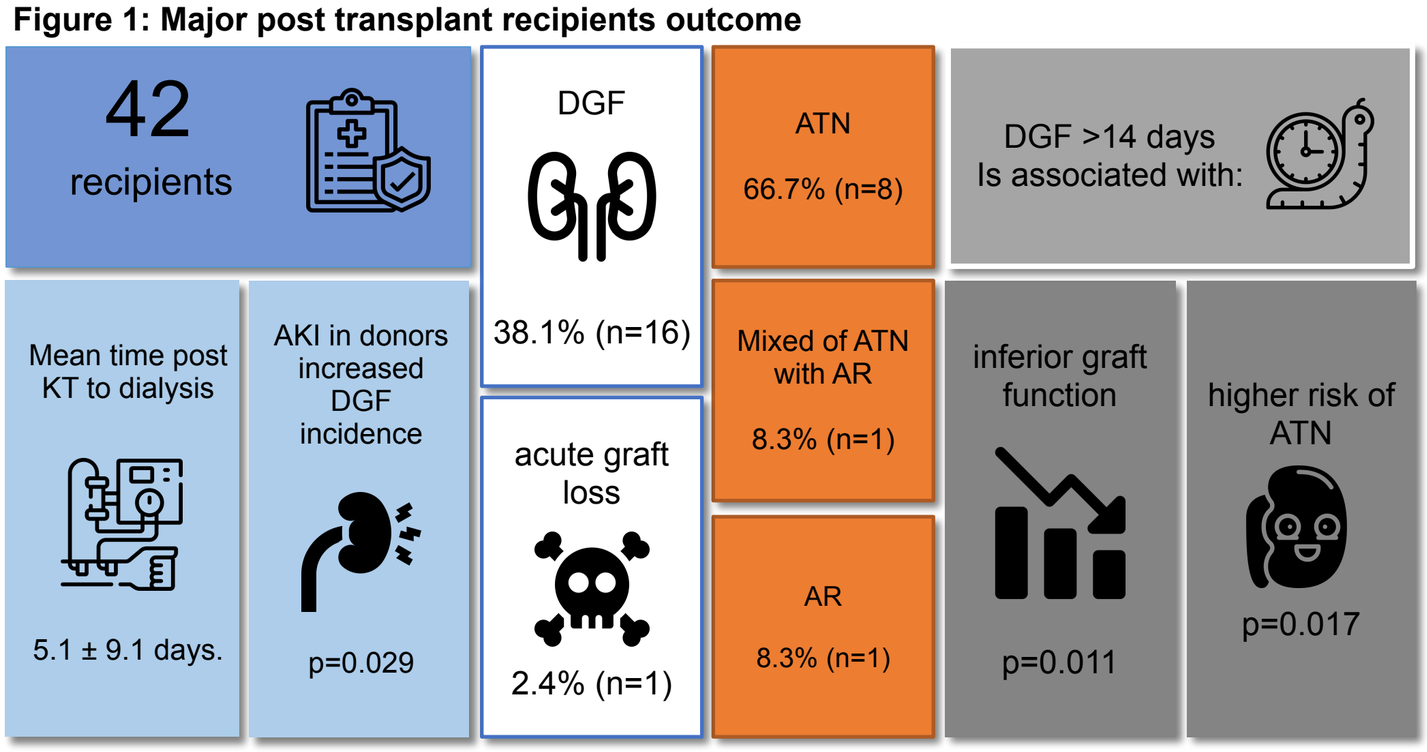
Results

Table 1: Donors and recipients demographics (n=42)

Recipient profile	
Male	19 (45.2%)
Female	23 (54.8%)
Mean age	38.3 ± 8.7 years
Mean dialysis vintage	13.9 ± 3.6 years
Mean Scr 3 months post KT	120.8 ± 47.9umol/l.

Donor profile	
Male	28 (66.7%)
Female	14 (33.3%)
Mean age	32.9 ± 9 years
Mean Scr on procurement	131.1 ± 91.6umol/l
Donor AKI	22 (52.4%)

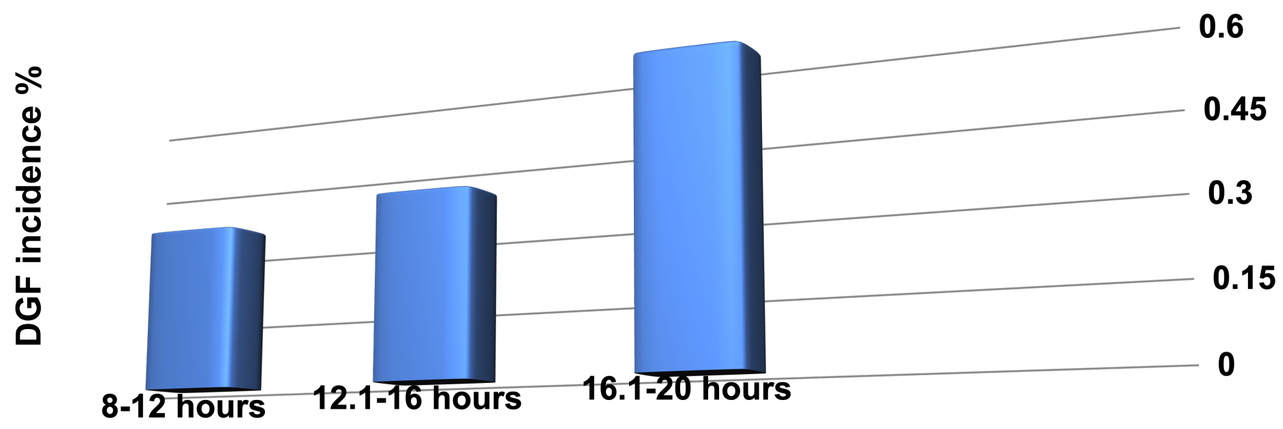
Figure 1: Major post transplant recipients outcome



Incidence of DGF was higher when CIT >16 hours.
8-12 hours: 33.3% (7 out of 21);
12.1-16 hours: 37.5% (6 out of 16);
16.1-20 hours: 60% (3 out of 5);
although statistically is not significant (p=0.44).

DGF >14 days was significantly associated with inferior graft function with eGFR <60 ml/min/1.73 m2 at 3rd month post KT (p = 0.011) and higher risk of ATN (p=0.017) but not to AR (p = 0.257) as shown in renal biopsy.

Figure 2: association of duration of CIT with DGF incidence



Discussion

Most of the studies have shown that AKI occurs in deceased donor prior to KT too frequently progresses to the clinical diagnosis of DGF[2]. Poor kidney function in the first week of graft life is detrimental to the longevity of the allograft[3]. To truly understand the root cause of DGF, protocol biopsies are needed in order to find out several pathologic contributors derived from either the donor or the recipient, commonly due to ischemic injury, inflammatory signaling, reperfusion injury, the innate immune response, and the adaptive immune response. Our cohort reported mainly ischemic injury associated with ATN as the main culprit of DGF based on the biopsies performed. Nevertheless, some studies showed that post transplant ATN does not appear to exert any detrimental effects in the long run. However, this issue remains controversial in the published reports.

Conclusion

In this cohort, CIT and AKI in donor upon procurement are associated with higher incidence of DGF. DGF showed significant short term inferior graft function from ATN. Longer study duration is needed to determine the effect of DGF on long term graft function. Measures to reduce CIT and careful selection of donors would mitigate DGF risk.

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