PHENOTYPE OF VASCULAR REJECTION AFTER KIDNEY TRANSPLANTATION: A HIDDEN THREAT

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Objectives:

Acute vascular rejection (AVR) is a severe clinical condition with detrimental impact on kidney allograft survival. Although it has been thought to be T-cell mediated process, recent clinical findings showed its association with donor specific alloantibodies and resistance to usual, against T-cells directed treatment. Therefore, correct assessment of AVR phenotype (T-cell mediated vs. antibody mediated) would be beneficial for adaptation of treatment strategy and improving long-term prognoses of kidney allografts.

Methods:

We retrospectively analysed 206 patients who underwent a kidney transplantation in year 2012. The incidence of AVR, risk factors, response to treatment, impact of rejection phenotype on graft function and survival was assessed. AVR was defined as a presence of intimal arteritis (v) in biopsies within 1 year after transplantation. "Isolated v-lesion" formed a subgroup of AVR and was characterized as an intimal arteritis with minimal interstitial inflammation and tubulitis (v1-3, i≤1, t≤1, C4d negat,, ptc negat., g negat.).

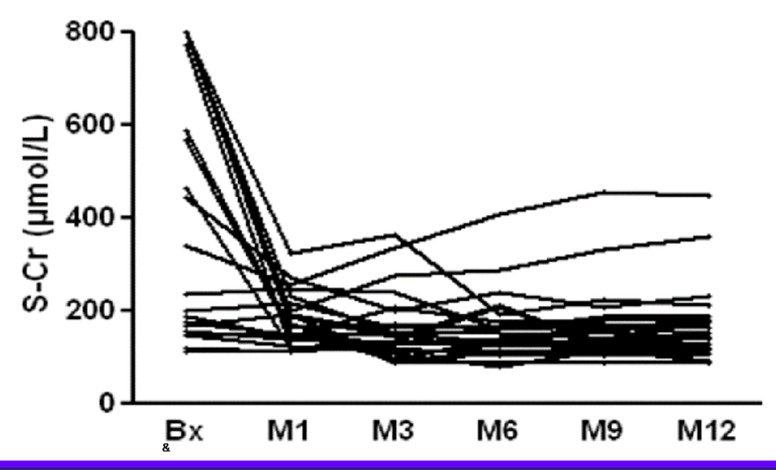

Fig. 1.
Renal function during 1 year of follow-up in patients with early acute vascular rejection.

Results:

AVR was found in 23/206 patients (11%), "isolated v-lesion" in 7/206 (3%). AVR represented 48% of all rejection findings within 1 year after Tx with median incidence of 24 days. In most cases (21/23, 91%) AVR was found in indication biopsies due to nonfunction or graft function deterioration. Only 2 cases were diagnosed from protocol biopsy and marked as subclinical "isolated v-lesion". Pathologist classified AVR as acute T-cell mediated rejection in 74% (TCMR). Remaining 26% cases were described as mixed T-cell and antibody-mediated rejection (AMR).

Conventional steroid treatment was applied in 11 (48%) patients. 55% biopsies were steroid-resistant and depletion (rATG) or B-cell targeted therapy had to be used. If rATG or AMR targeted therapy was used as initial treatment, v-lesions resolved in 75% and 84% patients, respectively.

Luminex was evaluated in 52% of AVR and confirmed to be positive in 75% of all evaluated cases. In 5 cases AVR was diagnosed after prior AMR. Chronic rejection developed in 4 cases, in 2 cases as a consequence of acute AMR and in other 2 cases as a result of steroid-resistant "isolated v-lesion".

Arteritis grade	**Patients** (n=23)
v1	15
v2	8
v3	0

Pathologist´s conlusion	**Patients** (n=23)
TCMR	17 (74%)
AMR	0
mixed AMR+ TCMR	6 (26%)

Table 1 and 2:
Histological grade of arteritis and pathologist´s conclusion based on currently Banff ´05 classification.

Primary treatment	**Patients** (n=23)	**V-lesion regression in surveillance biopsy&**	**Resistency to treatment**
steroids	11 (48%)	5 (45%)	6 (**55**%)
rATG	8 (35%)	6 (75%)	2 (25%)
AMR treatment (PP, IVIG)*	6 (26%)	5 (83%)	1 (16%)

Table 3
Response to primary treatment of vascular rejection
& No surveillance biopsy was performed in 4 patients.

Conclusions:

Vascular lesion is a frequent finding in the 1st year after renal transplantation and is associated with severe clinical course. Innovative diagnostic and therapeutic algorithms are essential to mitigate the impact of this rejection phenotype on transplanted kidney.

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