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Allo and auto crossmatches after transplantation

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The development of a positive donor-crossmatch after transplantation is usually seen as a bad prognostic sign with regard to graft survival. Since a number of positive post-transplant-crossmatches with the original donor in the absence of graft rejection was noticed in our center, we started a systematic investigation of donor-crossmatches after transplantation.

Key words: Kidney allografts – Crossmatches – Post-transplantation

Materials and methods

The development of T- and B-cell allo- and autoantibodies was investigated in 151 consecutive kidney allografts with a follow-up time of at least 1 year. Crossmatches were repeated with the original crossmatchsera as well as sera obtained after transplantation using frozen donor spleen cells and patients' peripheral blood lymphocytes. Crossmatches were performed using NIH and TCF techniques; DTT treatment was used to study the immunoglobulin class of the antibodies involved. Sera were collected at regular intervals and only patients with a minimum of three post-transplant sera were included in the study. The average number of post-transplant sera was six. Out of 151 patients, 100 had a negative donor crossmatch at the time of transplantation. Twenty-one grafts, performed with a positive T- and/or B-cell crossmatch due to autoantibodies, were excluded from the study, 19 patients had to be excluded for lack of material.

Results

After transplantation 60 patients formed neither allo- nor autoantibodies, 30 patients showed reactivity to donor B-cells but not T-cells and 20 patients developed antibodies reactive with donor T-cells (Table 1). Graft survival in the different groups was 90%, 80% and 75% respectively. The presence of autoantibodies was investigated in the different groups. From 20 patients with a positive T-cell donor crossmatch, 10 were shown to possess auto-T-cell antibodies and 2 auto-B-cell antibodies. In 30 patients with a positive B-cell donor crossmatch, autoreactivity could be shown in 13. One year graft survival in patients with autoantibodies was 84% in contrast to 72% in the group without autoantibodies.

Since graft survival seems to be influenced by the formation of T-cell antibodies, we selected the patients with good 1 year graft survival and no rejections to see whether this group lacked antibody development. Out of 54 patients, 15 developed a positive donor crossmatch after transplantation (13 B-cell, 2 T-cell) (Table 2). Out of 39 patients

Table 1. Donor crossmatches after transplantation and autoantibodies

	Donor crossmatch		
	T – B –	T – B +	T + B +
Auto crossmatch			
T – B –	60 (6)	17 (4)	8 (3)
T – B +	0	13 (2)	2
T + B +	0	0	10 (2)
	60	30	20

(⊕) = Grafts lost within one year

Table 2. Donor crossmatches after transplantation and rejection

		Donor crossmatch		
	N	T – B –	T – B +	T + B +
No rejection treatment	54	39	13 (6)	2 (1)
Reversible rejection	39	15	11 (5)	13 (9)
Irreversible rejection or graft loss	17	6	6 (2)	5 (2)

(⊕) = Auto crossmatch positive

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with reversible rejection, 24 became positive (11 B-cell, 13 T-cell) as well as 11 out of 17 patients with irreversible rejection or graft loss (6 B-cell, 5 T-cell). Of these positive donor crossmatches 50% were autocrossmatch positive.

All 7 patients who had been treated with OKT3 developed a positive donor-crossmatch after transplantation with strongly positive T + B + auto-crossmatches which reactivity could not be removed by DTT treatment. When the number of OKT3 treated patients was extended to 12, all of them (100%) showed a positive T-cell donor crossmatch after transplantation as compared to 3 out of 23 patients who received RATG as rejection treatment (13%). The sera of all treated patients were strongly cytotoxic as early as 24 h after the first administration of OKT3 and stayed positive until a maximum of 10 days after cessation of the therapy.

Conclusion

We concluded that the development of a positive T-cell donor crossmatch after transplantation influenced graft survival but was not necessarily a prediction of graft failure. A positive B-cell donor crossmatch did not seem to influence graft survival in patients with autoantibodies. Half of all positive donor crossmatches after transplantation were due to the development of autoantibodies. OKT3 caused strong cytotoxic activity of patient sera which could not be removed by DTT. Therefore sera collected during OKT3 treatment could not be used for crossmatching or antibody investigation.