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Desensitization Combined with Paired Exchange Leads to Successful Transplantation in Highly Sensitized Kidney Transplant Recipients: Strategy and Report of Five Cases

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Abstract

Sensitization remains a major barrier to kidney transplantation. Sensitized patients comprise 30% of the kidney transplant waiting list but fewer than 15% of highly sensitized patients are transplanted each year. Options for highly sensitized patients with an immunologically incompatible live donor include desensitization or kidney paired donation (KPD). However, these options when used alone may still not be sufficient to allow a compatible transplant for recipients who are broadly sensitized with cumulative calculated panel reactive antibody (cPRA) > 95%. We describe in this report the combined use of both desensitization and KPD to maximize the likelihood of finding a compatible match with a more immunologically favorable donor through a kidney exchange program. This combined approach was used in five very highly sensitized patients, all with cPRA 100%, who ultimately received compatible living and deceased donor kidney transplants. We conclude that early enrollment in paired kidney donor exchange and tailored desensitization protocols are key strategies to improve care and rates of kidney transplantation in highly sensitized patients.

Keywords

desensitization; kidney exchanges; kidney transplantation; HLA antibodies; donor-specific antibodies

Introduction

Kidney transplantation is the treatment of choice for patients with end-stage renal disease (ESRD) (1). Sensitization is a major barrier to successful kidney transplantation. Sensitized patients comprise approximately 30% of the deceased donor waiting list and have the

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longest waiting times because of difficulty in finding a compatible donor (2). Despite priority status in the organ allocation algorithm, fewer than 15% of highly sensitized patients are transplanted per year (3). Based on Organ Procurement and Transplantation Network (OPTN) data as of the end of 2010, although patients with cumulative calculated panel reactive antibody (cPRA) 80–95% have benefited with increased transplantation rates, those patients with cPRA > 95% remain difficult to transplant especially those fully sensitized with cPRA 100%. Current options for highly sensitized patients with an immunologically incompatible live donor include desensitization, kidney paired donation (KPD), or a combination (4).

For the past decade, desensitization has been successful in select patients. Current agents for desensitization include intravenous immunoglobulin (IVIG), rituximab, plasmapheresis, as well as newer agents such as bortezomib and eculizumab (5–7). Recent studies show a survival benefit that more than doubled by eight years for patients undergoing desensitization and transplantation compared with remaining on dialysis (8). Unfortunately, many patients do not respond to desensitization, especially highly sensitized patients with broad and strong human leukocyte antigen (HLA) antibody reactivity. Furthermore, long-term outcomes in positive crossmatch kidney transplant recipients may be inferior compared to immunologically compatible transplants (9). KPD is a creative option that matches a more immunologically compatible donor with a recipient through a registry. However, difficult-to-match donor-recipient combinations, including broadly sensitized patients, continue to pose a challenge.

Combining desensitization and KPD for patients who have strong antibody reactivity to a proposed but willing donor, while also keeping them on the deceased donor waiting list, increases the pool of potential donors. We hypothesized that this combined approach would enable our highly sensitized patients to be transplanted. Other centers, including Johns Hopkins, have combined KPD and desensitization to increase transplant rates (10–12). Both desensitization and KPD programs are expensive and require significant resources. Therefore, we sought to determine how best to utilize these strategies in highly sensitized patients.

We report our experience of five highly sensitized patients, all with cumulative cPRA 100%, who underwent desensitization in combination with KPD and successfully received kidney transplants. By carefully analyzing donor and recipient HLA typing and antibodies, our objective is to create a strategy on how to enroll donor-recipient pairs in KPD and prescribe desensitization therapy to enable successful transplantation in the very highly sensitized patients.

Case Histories

The recipient demographic information, HLA typing, histocompatibility data, and clinical outcomes are depicted (Tables 1–4). Desensitization therapy consisted of monthly high-dose intravenous immunoglobulin (IVIG) (2 gm/kg), rituximab (375 mg/m²) after four doses of IVIG, and plasmapheresis followed by bortezomib at 1.3 mg/m² in one patient who did not respond to IVIG and rituximab. Immunosuppression was anti-thymocyte globulin induction, IVIG (2 gm/kg) at the time of transplant followed by another dose three weeks post-transplant, and mycophenolate mofetil (MMF), tacrolimus, and prednisone for maintenance immunosuppression. The patients and their incompatible donors were entered into the National Kidney Registry (NKR).

An acceptable crossmatch to proceed with transplantation was T-cell and B-cell flow crossmatch (FXM) with a median flow-channel shift (MCS) of 200 after adjusting for presence of autoantibodies (normal range MCS T-cell FXM 88 and B-cell FXM 100).

HLA antibodies are considered positive with normalized mean fluorescence intensity (MFI) of ≥ 1000 and “possible” with MFI 500–999. All patients had post-transplant donor specific antibody (DSA) monitoring and protocol kidney biopsies at implantation and 3 and 12 months post-transplant. We performed retrospective T-cell and B-cell FXM and DSA analyses for recipients against both their original intended donors and KPD donors to elucidate the effect of desensitization and matching on ultimately finding a compatible donor (Table 3).

Case 1

The patient is a 33-year-old man with ESRD secondary to biopsy-proven primary focal segmental glomerulosclerosis (FSGS) (Table 1). He received a living kidney transplant with his mother as donor in 1995. He experienced acute cellular rejection in 1997, and his graft failed secondary to biopsy-proven recurrent FSGS and transplant glomerulopathy. He started peritoneal dialysis in 2008. He underwent transplant nephrectomy in 2009. In 2010 his friend (56-years-old, blood type O) came forward as a potential donor. Their FXM was strongly positive: T-cell 475 MCS and B-cell 477 MCS with several DSA (Table 3).

In an attempt to undergo a living kidney transplant with his friend as the intended donor, in March 2010, he started desensitization therapy with monthly high-dose IVIG infusions. In June 2010 he had partial response to therapy with T-cell 250 MCS and B-cell 320 MCS. Because he was still above the FXM cutoff to proceed with transplantation, the patient and his potential donor enrolled in KPD while continuing desensitization. In July 2010 a match from a 45-year-old, blood type O potential donor was identified in the KPD pool. The FXM against the donor in the exchange was T-cell 148 MCS and B-cell 245 MCS with a “possible” DSA to A2 (Table 3). He received a kidney transplant from this donor in July 2010. His protocol biopsy at 3 months post-transplant showed borderline acute cellular rejection (C4d negative) treated with prednisone (Table 4). He has not developed post-transplant DSA and has stable allograft function without proteinuria 24 months post-transplant.

Case 2

The patient is a 46-year-old man with ESRD secondary to biopsy-proven IgA nephropathy (Table 1). He received a deceased donor kidney transplant in 1993. He suffered two episodes of acute cellular rejection and subsequently started dialysis in 2007. His friend (48-years-old, blood type O) came forward as a potential donor. Their FXM was negative for T-cells (8 MCS) and positive for B-cells (288 MCS) mainly driven by a high-strength DSA to DQ2 (Table 3).

He began desensitization with monthly high-dose IVIG infusions in December 2009 and rituximab in March 2010. After four months of desensitization, the B-cell FXM remained strongly positive with a persistent antibody to DQ2 (Table 3). In June 2010 the patient and his potential donor entered into KPD while continuing desensitization. In August 2010 a match from a 64-year-old, blood type O, DQ2 negative, potential donor was identified in the KPD pool. The FXM against the donor in the exchange was T-cell 39 MCS and B-cell 228 MCS. He received a kidney transplant from this donor in August 2010. He has stable allograft function without DSA 23 months post-transplant (Table 4).

Case 3

The patient is a 30-year-old man with ESRD secondary to biopsy-proven lupus nephritis. He received a deceased donor kidney transplant in 1996 that failed in 2001 secondary to chronic rejection. He underwent a transplant nephrectomy in 2002. He also suffered from difficulties

with dialysis access and, eventually, was dialyzing through a right thigh arteriovenous fistula.

In March 2008 he started desensitization in an attempt to receive a compatible transplant from the deceased donor waiting list. He received monthly high-dose IVIG, rituximab (two doses), and plasmapheresis. In June 2010 his friend (24-year-old, blood type A) came forward as a potential donor. Their FXM was strongly positive: T-cell 383 MCS and B-cell 434 MCS with multiple high level DSA (Table 3). Because of strong reactivity against the intended donor, the patient and donor entered into KPD. In addition, he began therapy with plasmapheresis and bortezomib in combination with IVIG in an attempt to lower high-strength HLA antibodies (Figure 1). In August 2010, after three cycles of plasmapheresis and bortezomib, a match from a 33-year-old, blood type A potential donor was identified in the KPD pool. The FXM against the donor in the exchange was T-cell 171 MCS and B-flow 254 MCS with only one low positive DSA. He subsequently received a kidney transplant from this donor in September 2010. Post-transplant he developed a *de novo* DSA against Cw6. His protocol 3-month kidney biopsy showed borderline acute cellular rejection (C4d negative) treated with prednisone. He remains with excellent graft function 22 months post-transplant (Table 4).

Case 4

The patient is a 33-year-old woman with ESRD secondary to cortical necrosis after meningococcal sepsis (Table 1). She received a pediatric en bloc deceased donor kidney transplant in 1999. She experienced acute cellular rejection in 2001 and vesiculoureteral reflux requiring ureter reimplantation. She underwent transplant nephrectomy 2002 at which time she returned to dialysis. In 2009 her mother (54 years-old, blood type O) came forward as a potential donor. Their FXM was positive: T-cell 399 MCS and B-cell 361 MCS.

She started monthly IVIG infusions in March 2010 with one dose of rituximab at that time. In June 2010 she had partial response to therapy with T-cell 327 MCS and B-cell 325 MCS. At that time the patient and her potential donor enrolled in KPD while continuing desensitization. In September 2010 she received a zero-mismatched deceased donor kidney transplant. She has stable allograft function without DSA 22 months post-transplant (Table 4).

Case 5

The patient is a 26-year-old man with ESRD secondary to obstructive uropathy from posterior urethral valves. He underwent a living related kidney transplant with his mother as a donor in 1998. He had several episodes of acute rejection and his transplant failed in 2004. He underwent transplant nephrectomy in 2009. In December 2010 his friend (22 years-old, blood type A) came forward as a potential donor. Their FXM was positive with T-cell 439 MCS and B-cell 464 MCS.

He began desensitization therapy with monthly IVIG infusions in May 2011 and rituximab October 2011. He had partial response to therapy. In an attempt to further decrease HLA antibodies, he proceeded with plasmapheresis in April 2012. After one session of plasmapheresis and prior to receiving bortezomib, he received an offer for a deceased donor transplant in April 2012. He has not developed post-transplant DSA after 3 months.

Discussion

We identified five highly sensitized kidney transplant recipients, all with cPRA 100%, who underwent desensitization prior to participating in KPD. We enrolled two patients in KPD after desensitization failed to lower high-strength HLA antibodies against the intended

donors. KPD enabled them to find compatible donors for whom they had low-strength or no HLA antibodies *prior* to desensitization. For patient 1, we found a donor lacking B44 for which the patient had strong reactivity. Although the patient had an antibody against A2, the MFI was low enough after desensitization therapy to proceed with transplantation. For patient 2, because the patient had strong reactivity to DQ2, which did not decrease with desensitization, we attempted to find a donor lacking DQ2. In addition, after desensitization, two low-strength DSA decreased. This approach was possible because these two patients were not broadly sensitized to most of the common HLA genotypes.

For patient 3, who had high-strength antibodies against common HLA antigens, desensitization was necessary to enable us to lower HLA antibodies sufficiently to find a compatible match from the KPD pool. We tested his serum prior to desensitization against the matched donor. Notably, the initial FXM showed strong reactivity to the donor in the KPD pool that diminished after desensitization. In this case, bortezomib seemed to have the most significant effect on lowering HLA antibodies (Figure 1) in contrast to published experience that did not find a beneficial desensitization effect with bortezomib (13). Perhaps the difference may have been that we used bortezomib in conjunction with IVIG, rituximab, and plasmapheresis in a manner similar to investigators who used bortezomib successfully in the setting of desensitization in heart transplantation (14).

Highly sensitized patients (cPRA ≥ 80%) have low match rates in KPD, often below 15% (15, 16). Most of these patients are sensitized to common HLA antigens and are seeking to match a donor with a rare HLA genotype. By computer simulations, we can predict who can find a match in a KPD database (15, 16). Predicting who responds to desensitization therapy is more challenging. Patients 4 and 5 are two highly sensitized patients, both blood type O, who underwent desensitization, enrolled in KPD, and subsequently received deceased donor kidney transplants. One intended donor was over 50 years old (blood type O) and the other intended donor was blood type A. In these two recipients who did not find a match in the KPD pool, desensitization enabled them to receive deceased donor kidney transplants. One patient received a zero-mismatched kidney from a deceased donor, and one patient received a compatible deceased donor.

Another strategy we and other centers, including Johns Hopkins, have implemented is to raise the threshold for listing “unacceptable antigens” in highly sensitized patients undergoing desensitization and, thereby, allow the presence of low-strength DSA (11). It is unclear whether low-strength HLA antibodies lead to allograft injury (17). Our center threshold for listing unacceptable HLA antigens is 1000 MFI. However, in patients undergoing desensitization, we list HLA antibodies with MFI strength 3000 or higher. Other centers have expanded the KPD pool by encouraging compatible pairs to participate, especially pairs with blood group O donors (18–20).

In these five highly sensitized patients, we used multiple strategies to increase their chances of undergoing transplantation. In each case, the patient’s potential living donor pool was expanded by enrolling them in a KPD program while increasing the number of potential compatible matches on the deceased donor list through desensitization. All five patients were successfully transplanted: three with living donors through KPD and two with deceased donors. Upon review of the changing levels of recipient HLA antibodies and the actual donor HLA genotypes, one recipient was able to find a donor through KPD only because he also underwent desensitization. Although the other recipients were transplanted through KPD or with a compatible deceased donor that would not have required them to be desensitized, the recipient HLA antibody profile against these grafts improved by desensitization. Therefore, early enrollment in KPD, participation in desensitization protocols, and implementation of less strict criteria to allow for presence of low-strength

DSA are viable strategies aimed to improve care and rates of transplantation in highly sensitized patients.

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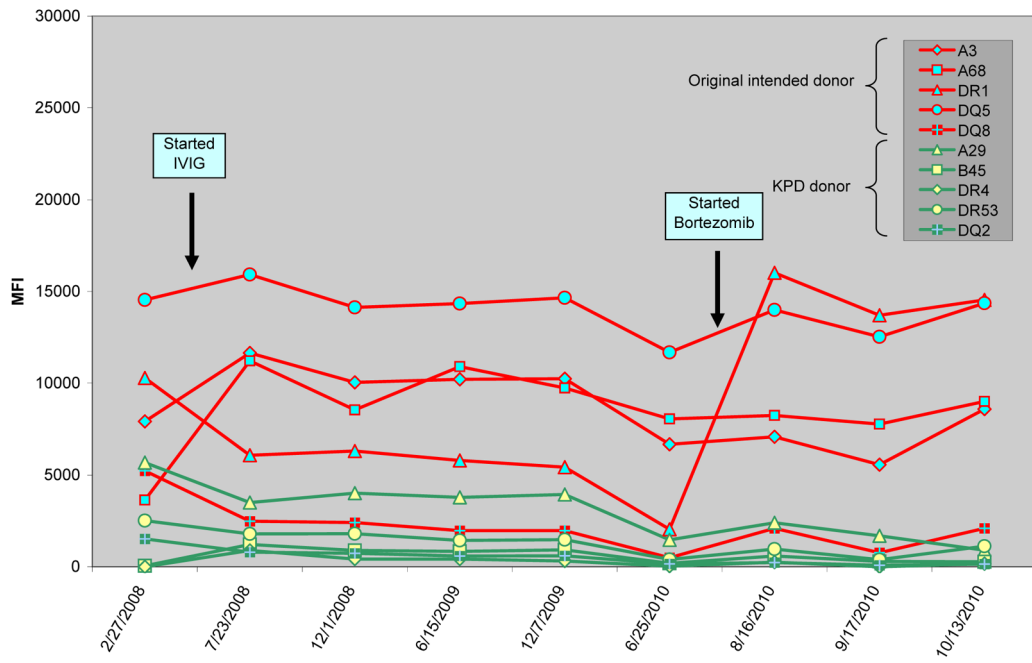


Figure 1. Effect of intravenous immunoglobulin (IVIg) and bortezomib on the donor specific antibodies (DSA) in both the original intended donor (red) and the KPD donor (green) in case 3.

Table 1

Patient demographics

Parameters	Case 1	Case 2	Case 3	Case 4	Case 5
Age at time of transplant (yr)	33	46	30	33	26
Sex	M	M	M	F	M
Cause of ESRD	FSGS	IgA	SLE	Cortical necrosis	Obstructive nephropathy
Prior kidney transplantation (n)	1	1	1	1	1
Waiting time for a transplant (yr)	3	5.5	8	8.2	5.2
Blood Type	O	O	A	O	O

FSGS = focal segmental glomerulosclerosis

IgA = IgA nephropathy

SLE = systemic lupus erythematosus

Table 2

HLA Typing

	HLA-A	HLA-B	HLA-Cw	HLA-DR	HLA-DQ
Case 1	A3, X	B65, 57	Cw8, 17	DR7, X	DQ2, 9
Original donor	A2, 29	B44, X	Cw16, 5	DR11, 15	DQ7, 6
KPD donor	A2, 26	B62, 38	Cw9, 12	DR4, 13	DQ8, 6
	HLA-A	HLA-B	HLA-Cw	HLA-DR	HLA-DQ
Case 2	A11, 26	B27, 35	Cw1, 4	DR1, 1	DQ5, X
Original donor	A1, 24	B8, 46	Cw1, 7	DR17, 14	DQ2, 5
KPD donor	A3, 30	B71, 55	Cw7, 9	DR13, 15	DQ6, 6
	HLA-A	HLA-B	HLA-Cw	HLA-DR	HLA-DQ
Case 3	A23, 26	B65, 35	Cw4, 8	DR8, 13	DQ4, 7
Original donor	A3, 68	B35, 39	Cw4, 7	DR1, 4	DQ5, 8
KPD donor	A23, 29	B44, 45	Cw4, 6	DR4, 7	DQ2, X
	HLA-A	HLA-B	HLA-Cw	HLA-DR	HLA-DQ
Case 4	A3, 29	B7, 7	Cw7, 15	DR13, 15	DQ6, 6
Original donor	A3, 68	B7, 44	Cw7, 7	DR13, 15	DQ6, 6
Deceased donor	A3, X	B7, X	Cw7, X	DR15, X	DQ6, X
	HLA-A	HLA-B	HLA-Cw	HLA-DR	HLA-DQ
Case 5	A3, 11	B8, 48	Cw7, 8	DR1, 11	DQ5, 7
Original donor	A2, 26	B62, X	Cw4, 9	DR9, 13	DQ6, 9
Deceased donor	A1, 30	B8, 62	Cw7, 10	DR1, 17	DQ2, 5

Table 3

Histocompatibility data.

I. Original intended donor									
Patient	Cum cPRA	Flow Cross match				DSA			
		pre-desensitization T cell XM auto/allo	pre-desensitization B cell XM auto/allo	post-desensitization T cell XM auto/allo	post-desensitization B cell XM auto/allo	pre-desensitization DSA (MFI)	post-desensitization DSA (MFI)	pre-desensitization DSA (MFI)	post-desensitization DSA (MFI)
Case 1	100%	NA/475	NA/477	112/250	134/320	A2 (913-1780) A29 (613-758) B44 (6867-9262)	A2 (571-922) A29 (501-561) B44 (6882-7972)	A2 (913-1780) A29 (613-758) B44 (6867-9262)	A2 (571-922) A29 (501-561) B44 (6882-7972)
Case 2	100%	NA/8	NA/288	50/63	27/338	A24 (1154-1582) DQ2 (15799-19749)	A24 (Neg) DQ2 (19304-21311)	A24 (1154-1582) DQ2 (15799-19749)	A24 (Neg) DQ2 (19304-21311)
Case 3	100%	98/383	177/434	NA	NA	A3 (13068) A68 (9535-9931) DR1 (5855-6021) DQ5 (8761-14004) DQ8 (849-1290)	A3 (5696) A68 (9660-9204) DR1 (16942-17523) DQ5 (13911-15814) DQ8 (175-1319)	A3 (13068) A68 (9535-9931) DR1 (5855-6021) DQ5 (8761-14004) DQ8 (849-1290)	A3 (5696) A68 (9660-9204) DR1 (16942-17523) DQ5 (13911-15814) DQ8 (175-1319)
Case 4	100%	NA/399	NA/361	30/327	24/325	A68 (5445-6209) B44 (15137-15455)	A68 (5220-5652) B44 (9750-9790)	A68 (5445-6209) B44 (15137-15455)	A68 (5220-5652) B44 (9750-9790)
Case 5	100%	17/439	50/464	NA	NA	A2 (528-1467) A26 (1165) B62 (2044) DR53 (7181-9685) DQ9 (669)	A2 (1543-2209) A26 (757) B62 (556) DR53 (7493-9862) DQ9 (Neg)	A2 (528-1467) A26 (1165) B62 (2044) DR53 (7181-9685) DQ9 (669)	A2 (1543-2209) A26 (757) B62 (556) DR53 (7493-9862) DQ9 (Neg)

II. KPD donor or Deceased donor									
Patient	Cum cPRA	Flow Cross match				DSA			
		pre-desensitization T cell XM auto/allo	pre-desensitization B cell XM auto/allo	post-desensitization T cell XM auto/allo	post-desensitization B cell XM auto/allo	pre-desensitization DSA (MFI)	post-desensitization DSA (MFI)	pre-desensitization DSA (MFI)	post-desensitization DSA (MFI)
Case 1	100%	NA	NA	111/148	124/245	A2 (913-1780)	A2 (571-922)	A2 (913-1780)	A2 (571-922)
Case 2	100%	NA	NA	48/39	47/228	DR13 (591-1755) DQ6 (805)	DR13 (Neg) DQ6 (Neg)	DR13 (591-1755) DQ6 (805)	DR13 (Neg) DQ6 (Neg)
Case 3	100%	243/325	257/324	130/171	148/254	A29 (3314-3967) B45 (899) DR4 (441-965) DR53 (1435-2373) DQ2 (473-1935)	A29 (1279-1498) B45 (Neg) DR4 (Neg) DR53 (552-706) DQ2 (Neg)	A29 (3314-3967) B45 (899) DR4 (441-965) DR53 (1435-2373) DQ2 (473-1935)	A29 (1279-1498) B45 (Neg) DR4 (Neg) DR53 (552-706) DQ2 (Neg)
Case 4	100%	NA	NA	46/0	30/14	None	None	None	None
Case 5	100%	NA	NA	0/40	1/2	B62 (2044)	B62 (556)	B62 (2044)	B62 (556)

Table 4

Clinical outcomes

Parameters	Case 1	Case 2	Case 3	Case 4	Case 5
Post-operative (mo)	24	23	22	22	3
Acute rejection					
Borderline	1	0	1	0	0
Cell-mediated	0	0	0	0	0
Antibody-mediated	0	0	0	0	0
Creatinine (mg/dl) 1 mo	1.5	1.4	1.6	1	2.5
Creatinine (mg/dl) 3 mo	1.6	1.4	1.5	1	2.1
Creatinine (mg/dl) 6 mo	1.8	1.6	1.3	1	N/A
Creatinine (mg/dl) 1 yr	1.4	1.4	1.5	0.9	N/A
Last creatinine (mg/dl) post-transplant (mo)	1.5 (24)	1.5 (23)	1.1 (22)	0.9 (22)	2.1 (3)
Graft loss (%)	0	0	0	0	0