Paired Kidney Donor Exchanges and Antibody Reduction Therapy: Novel Methods to Ameliorate Disparate Access to Living Donor Kidney Transplantation in Ethnic Minorities

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BACKGROUND: Currently ethnic minority patients comprise 60% of patients listed for kidney transplantation in the US; however, they receive only 55% of deceased donor renal transplants and 25% of living donor renal transplants. Ethnic disparities in access to kidney transplantation result in increased morbidity and mortality for minority patients with end-stage renal disease. Because these patients remain dialysis dependent for longer durations, they are more prone to the development of HLA antibodies that further delay the possibility of receiving a successful kidney transplant.

STUDY DESIGN: Two to 4 pretransplant and post-transplant plasma exchanges and IV immunoglobulin were used to lower donor-specific antibody levels to less than 1:16 dilution; cell lytic therapy was used additionally in some cases. Match pairing by virtual cross-matching was performed to identify the maximal exchange benefit. Sixty candidates for renal transplantation were placed into 4 paired kidney exchanges and/or underwent antibody reduction therapy.

RESULTS: Sixty living donor renal transplants were performed by paired exchange pools and/or antibody reduction therapy in recipients whose original intended donors had ABO or HLA incompatibilities or both (24 desensitization and 36 paired kidney exchanges). Successful transplants were performed in 38 ethnic minorities, of which 33 were African American. Twenty-two recipients were white. Graft and patient survival was 100% at 6 months; graft function (mean serum creatinine 1.4 g/dL) and acute rejection rates (20%) have been comparable to traditional live donor kidney transplantation.

CONCLUSIONS: Paired kidney donor exchange pools with antibody reduction therapy can allow successful transplant in difficult to match recipients. This approach can address kidney transplant disparities. (J Am Coll Surg 2011;212:740–747. © 2011 by the American College of Surgeons)

Currently in the field of kidney transplantation, there is an increasing crisis between those patients who would benefit from a kidney transplant and the availability of a life-saving organ. More than 85,000 individuals are listed for kidney transplantation, and only approximately 16,000 patients receive life-saving transplants every year. This crisis is keenly felt in the minority communities of the US both because the incidence of end-stage renal disease (ESRD) is markedly increased among several ethnic groups and access to organ transplantation has been a challenge.[1] Not only has access to renal transplantation been an issue for ethnic minority patients, but also outcomes have traditionally been worse than those for their majority counterparts after successful kidney transplantation.[2] The primary culprit in compromising both access to kidney transplantation and outcomes after transplantation may be related to the length of time minority patients with ESRD spend receiving dialysis treatments.[3]

Minority patients, particularly African-American (AA) patients, remain on hemodialysis markedly longer than all other patient groups, and this may lead to worse transplant

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Abbreviations and Acronyms
AA = African American
DSA = donor-specific antibodies
ESRD = end-stage renal disease
PKDE = paired kidney donor exchange

outcomes because of accelerated cardiovascular and vascular disease, increased incidence of infectious complications, and other physiologic perturbations.[4] Of special note is the fact that patients who remain hemodialysis dependent for long periods of time seem to be much more prone to the development of anti-HLA antibodies.[5] Once a prospective transplant patient becomes sensitized, matching for a suitable donor organ becomes much more difficult. For many of these patients, the likelihood of receiving a deceased donor organ via our current national matching algorithm is lessened.

The obvious solution to this crisis is to decrease the length of time on dialysis and perform more living donor renal transplants in this population of patients. There are many hurdles to increasing the rate of living donor renal transplantation in this patient population; however, the primary problem is the availability of matched living kidney donors.[6]

Our philosophy has been to attempt to use all available technology (desensitization, paired kidney donor exchanges, ABO-incompatible kidney transplantation, virtual cross-matching, use of nondirected kidney donors) to perform more living donor kidney transplants for all of our patients but particularly minority patients. Traditionally these inventive transplant strategies have not been used often in minority patients, and we have been strident in our belief that this patient population with the greatest need for living kidney transplantation should attract the methods that would allow for an increased rate of transplantation in their community. It is obvious that minority patients benefit more from living donor renal transplantation because this type of kidney transplantation markedly decreases their transplant waiting list time. In comparison with other ethnic groups, their outcomes from living donor renal transplantation vastly outstrips their outcomes from deceased donor renal transplantation.[7] Fewer than 15% of living donor renal transplants are performed in AA recipients nationally, and we have sought with our program to try to increase the distribution of living donor kidneys in this group.

METHODS

From September 2008 to June 2010, 4 distinct paired kidney exchanges as well as antibody reduction therapy (desensitization) were performed to achieve 60 living donor renal transplants. Etiologies of renal failure included hypertension (n = 18), diabetes (n = 14), glomerulonephritis (n = 7), lupus (n = 5), polycystic kidney disease (n = 5), IgA nephropathy (n = 3), congenital renal failure (n = 3), polyarteritis nodosa (n = 1), thrombotic thrombocytopenic purpura (n = 1), tacrolimus toxicity after intestinal transplant (n = 1), and idiopathic (n = 2).

There were 22 whites, 33 AAs, and 5 other ethnic minorities. Thirty-nine of our patients were ABO incompatible or highly sensitized. Forty-five of the recipients had their own donors, but they were incompatible. There were 7 nondirected donors and 1 zero antigen mismatch deceased donor. Virtual cross-matching was then used to match potential recipients and donors to facilitate the best match and elucidate which patients might require desensitization treatments. Recipients who were able to receive transplants from their intended donors were slated to receive their desensitization treatments and have their transplant operations. If the recipients could not be matched with their intended donors, then this group of patients was placed into a pool of patients for a paired kidney donor exchange (PKDE).

Induction therapy with T-cell–depleting antibodies was used in all patients. Adequacy of lymphocyte depletion was followed postoperatively by absolute CD3 counts. Maintenance therapy consisted of tacrolimus and mycophenolic acid with or without prednisone. In highly sensitized patients, 2 to 4 pretransplant and posttransplant plasma exchanges and IV immunoglobulin were used to lower donor-specific antibodies (DSA) to a titer less than 1:16. In addition, cell lytic therapy was used in some cases.

There was a rapid steroid wean in the 21 patients who were neither ABO incompatible nor highly sensitized. Highly sensitized and ABO-incompatible patients were maintained on prednisone 20 mg for the initial phase of maintenance therapy. IV ganciclovir was given for initial cytomegalovirus prophylaxis and then switched to valganciclovir.

Protocol biopsies were obtained at 1, 3, and 12 months. DSA titers were obtained weekly for the first month and then monthly for 1 year. Clinical indication for a biopsy also included a 2-fold increase in DSA and a rise in serum creatinine by 15%. All biopsies were stained with hematoxylin and eosin for immunofluorescent analysis of C4d.

Biopsies with cell-mediated rejection were treated according to severity of rejection. Banff grade 1 rejection was treated with steroid taper, with nonresponders receiving lymphocyte depletion therapy. Banff grades 2 and higher received T-cell depletion therapy. All humoral-mediated rejection was treated with plasmapheresis and IV immunoglobulin with or without bortezomib.

Table 1. Patient Demographics and Outcomes

Category	White	AA	Other	Mean creatinine, mg/dL	Median creatinine, mg/dL	Range of creatinine, mg/dL	Patient/graft survival
Non-PKDE							
ABO incompatible	7	3	0	1.38	1.3	0.9–2.1	100%/100% (12 mo)
Sensitized	5	4	1	1.34	1.3	0.9–2.3	100%/100% (9.9 mo)
PKDE							
ABO incompatible	2	4	0	1.58	1.35	0.8–2.6	100%/100% (6.4 mo)
ABO compatible nonsensitized	6	12	3	1.44	1.4	0.8–3.2	100%/100% (4.4 mo)
ABO compatible sensitized	2	10	1	1.38	1.3	0.9–1.9	100%/100% (7.4 mo)
Total	22	33	5	1.42	1.3	0.8–3.2	100%/100% (7.4 mo)

AA, African American; PKDE, paired kidney donor exchange.

If rebound of DSA titers above 1:16 were noted, patients had additional treatment with plasmapheresis and IV immunoglobulin. Variables analyzed included graft and patient survival, rejection episodes, and mean, median, and range of creatinine levels.

We analyzed our outcome data for the last 4 years to determine what impact our new program has had on availability of living donor kidney transplants for our patients. The Fisher exact test was used to analyze discrete valuation. $p < 0.05$ was deemed significant.

RESULTS

Multivariate approach used to foster living donor renal transplants

We performed 60 (10 sensitized, 10 ABO-incompatible, and 40 PKDE patients) living donor renal transplants in this program. Forty-five recipients had their own but incompatible donors. Sixty-five percent of these patients were from ethnic minorities (50% AA and 15% other minorities). In our cohort, 39 of the 60 patients (65%) transplanted were either highly sensitized or ABO incompatible. Thirty-eight of our recipients (63%) were from ethnic minorities, 33 of whom were AA (55%). Twenty-three of the 39 sensitized or ABO-incompatible patients (58%) were from ethnic minorities. These patients represent that subset of patients who otherwise may not have been transplanted. All patients received a transplant within 90 days of their initial evaluation for living donor transplantation.

At 7.4 months' follow up, the mean serum creatinine was 1.42 mg/dL, with (1-year) patient and graft survival of 100%. No significant differences were noted among the different groups (Tables 1–3).

Posttransplant biopsies were obtained by protocol in highly sensitized and ABO-incompatible recipients and others as deemed necessary. There was an 18% rate of diagnostic acute rejection and an additional 12% when biopsies suspicious but not diagnostic for acute rejection were included. Specifically among ABO-incompatible recipients (16 patients), 4 patients had an episode of diagnostic acute rejection. Two patients had a single episode of rejection (Banff grades 1A and 1B), 1 patient had 2 episodes of rejection (1A and 1B), and another patient had 3 episodes of rejection (1A, 1B, and 2A). There were also 2 patients with biopsies that were suspicious but not diagnostic for acute rejection. Among sensitized patients (23 patients), there were 5 patients with a single episode of diagnostic acute rejection (1A, 1A, 1A, mild, and 2A) and 2 patients with biopsies suspicious but not diagnostic for acute rejection. In the nonsensitized patients (21 patients), there were 2 patients with a single episode of diagnostic acute rejection (1A and 1B) and 3 patients with biopsies that were suspicious but not diagnostic for rejection. This is comparable to traditional living donor kidney transplantation.

PKDE program has led to increased rate of transplantation

We reviewed the scientific registry for transplant recipients to assess the impact of the new program. We performed 48, 32, 41, and 74 kidney transplants in 2006, 2007, 2008, and 2009, respectively. During this time, we performed 16, 12, 23, and 46 living donor transplants. This increased number of living donor transplants performed each year tangentially reflects a striking rise in the waiting list transplant rate.

Moreover, 232, 241, 237, and 231 patients were on the waiting list in the respective years leading up to the end of 2009. Strictly considering this "end of year" waiting list

Table 2. Demographics of Local Paired Kidney Donor Exchange Participants Versus National Living Donor Kidney Recipients

Category	Local (PKDE), %	US, %
African American	55	13.7
White	37	66
Other	8	20
Male	50	61
Female	50	39

PKDE, paired kidney donor exchange.

Table 3. Recipient Characteristics

Recipient demographics (n = 60)	
Race, n	
White	22
African American	33
Other	5
Clinical characteristics, n	
Previous kidney transplant	17
Diabetes	14
Hypertension	18
Polycystic kidney disease	5
Glomerulonephritis	7
Lupus	5
IgA nephropathy	3
Idiopathic	2
Other	6
Panel reactive antibody, %	
0–9	29
10–49	13
50–79	6
>80	10
Unknown	2
Time on hemodialysis, n	
<1 y	9
1–3 y	18
3–5 y	7
>5 y	5
Peritoneal dialysis	9
No dialysis	8
Unclear length of time	4
ABO incompatible, n	16
Sensitized, n	24
Recipient age, y	21–66

patient count, the germination of the PKDE program has increased the rate of kidney transplantation almost 2-fold. In 2006, 2007, and 2008, the overall rate of waiting list kidney transplantation was 20.7% ($p = 0.006$), 13.3% ($p = 0.0001$), and 17% ($p = 0.0002$), respectively, whereas the rate increased to 32% in 2009 after the introduction of the program.

Moreover, the PKDE program also increased the living donor kidney transplant rate among our waiting list. The living kidney donor waiting list rate of transplantation was 6.9% ($p = 0.0001$) in 2006, 5% ($p = 0.0001$) in 2007, 9.7% ($p = 0.0025$) in 2008, and 19.9% in 2009.

In summary, in 2006 through 2008, AAs underwent 32 deceased donor transplants and 18 living donor transplants. Interestingly in 2009, AAs underwent 12 deceased donor transplants and 17 living donor transplants. Although this reversal of the ratio of deceased donor:living donor kidney transplantation was significant ($p = 0.05$), it was suggestive of increased rates of kidney transplantation in AAs because of increased living donor transplantation. Moreover, this finding was likely driven by the PKDE program, which markedly increased living donor transplantation performed at our institution.

This observed rise is also noticeably apparent in the increased yearly rate of waiting list transplantation in AAs. The AA living kidney donor waiting list rate of transplantation was 3% ($p = 0.04$), 1.7% ($p = 0.003$), 3% ($p = 0.04$), and 7.4% in 2006 through 2009, respectively. These differences were significant when compared with 2009. Additionally, the overall AA waiting list rate of transplantation was significantly increased in 2009 as compared with that in 2006 through 2008. In summary, in 2006 through 2008, a transplant rate of 7% was achieved among AAs, whereas it was 12.6% in 2009. This almost 2-fold rise in transplantation rate was significant ($p = 0.01$).

DISCUSSION

PKDE and antibody reduction protocols offer unique new opportunities to increase successful kidney transplantation.[8-10] In many instances, candidates for renal transplantation have willing donors who, because of ABO or HLA incompatibility, are deemed unsuitable for transplantation to the intended recipient. Historically, the recipients would then go on the deceased donor waiting list and linger for 5 to 7 years in many areas before they were able to reach the top of the list for a suitable kidney for transplantation.

Minorities in particular face daunting challenges owing to disparities in referral and access, as well as biologic inequities in the donor population. Thus, the waiting times for minorities are significantly longer than for their majority counterparts. Because minorities wait longer and in most instances are referred after initiating dialysis, they are also more often sensitized through exposure to HLAs producing antibodies against DSA. Additionally because of the high prevalence of diabetes, hypertension, and obesity in minority populations, in many instances there are limited living donor opportunities in the circumstance of an incompatible suitable donor.[11] Recognizing these inherent disadvantages to minorities gaining access to successful kidney transplantation, we initiated a combination of novel strategies to successfully transplant individuals awaiting kidney transplantation to address the issue of prolonged waiting times and to determine if these novel therapeutic protocols could be applied to a large minority kidney waiting list. Thus, we initiated a program of PKDE and HLA desensitization between 2 urban transplant centers with large numbers of minority candidates on the respective waiting lists to improve access to live kidney transplantation.

Racial disparities in the allocation and outcomes of kidney transplantation in AAs have been well documented.[12-15] The markedly increased prevalence of diabetes and hypertension in AAs is responsible for an increased incidence of ESRD leading to hemodialysis in this group.[16] As a result, compared with whites, AAs have a 4-fold increased rate of developing ESRD.[17]

Interestingly, although AAs account for 37% of patients with ESRD, they receive only 25% of deceased donor kidneys in the US.[18] In examining this racial disparity, several observations have been made by the epidemiologic community. Epstein and colleagues[12] conclusively showed that there are delayed referral patterns for transplantation in appropriate candidates among AAs. Unfortunately, this practice has led to a higher degree of presensitizaton secondary to increased pretransplantation dialysis times.[19,20] As a consequence of delayed referral patterns, longer time periods of hemodialysis raise the level of HLA sensitization, which further increases time to transplant. Furthermore, time on hemodialysis has been shown to be a significant predictor of mortality.[21]

Other factors that hinder transplantation in the AA community are HLA polymorphisms coupled with current immunologic matching criteria that may stymie unbiased kidney allocation.[22] Lastly, there seems to be a shortage of AA living kidney donations.[23] This lack of representation for living donor donation in many instances is not due to lack of interest but rather the inherent prevalence of comorbidities in the minority population that limits the potential number of medically suitable compatible candidates for transplantation.

In an attempt to redress the unbalanced nature of kidney transplantation, the PKDE and antibody reduction program was established at our 2 urban transplant centers. In comparing the 4 successive years at our centers, the PKDE program dramatically increased the number of AAs transplanted. More importantly, the percentage of AA waiting list candidates transplanted in the year after initiation of the program increased to 12.6% in 2009. This 2.5-fold increase over previous years represents a distinct change in the waiting list transplant rate.

Although it is obvious that this rate of growth was accomplished with living donor transplantation, the current observations reported in our data counter the argument of a lack of "willingness" to donate in the AA community.[23] Moreover, our results further call into question the perception that financial concerns, fear of surgery, and distrust of the medical establishment are significant barriers to living donor donation.[24] In contrast, the PKDE program suggests that perhaps the low number of AA living donors is not a result of desire but of the immunologic pressures aforementioned.

The PKDE and antibody reduction program has created a culture within our program such that a recipient who has a willing medically suitable donor will receive a live donor kidney transplant through inclusion in one or more of our protocols, even if they are ABO or HLA incompatible. As such, we have changed our fundamental approach to kidney transplantation and exploit all tools available for induction therapy. Moreover, ABO-incompatible donor transplantation and desensitization protocols have become central pillars of our PKDE program.

ABO-incompatible kidney transplantation gained popularity in the 1990s and has demonstrated favorable long-term graft survival.[25] Initially characterized by A2 living donation to B or O recipients,[26-28] Japanese investigators spearheaded non-A2 donation, which has also shown equivocal outcomes.[29] Desensitization has provided a detour around high anti-A2 antibodies as well as other DSA.

Desensitization regimens that reduce DSA either through plasmapheresis or B-cell depletion have been increasingly used for induction.[25,30] Our strategy was patterned after Montgomery and colleagues,[31] who demonstrated 88.7% 5-year graft survival and function.

Our PKDE program employed desensitization protocols to treat 39 patients, which represented 65% of the patients involved in our exchanges. To date, graft survival is 100%, with a mean creatinine of 1.4 g/dL. Although these approaches are not novel, they have facilitated the expansion of participants by circumventing previously held limitations to transplantation. More notably, these expanded criteria have resulted in an overall increase in kidney transplantation at our center. The year 2009 was marked by a 32% rate of overall waiting list kidney transplantation, which was significantly higher than that in previous years.

Ultimately, our program was conceived out of a need to improve parity of organ distribution in an urban environment and to include a large number of minorities who may benefit the most from these novel approaches given the difficulties in obtaining successful live donor kidney transplants. In doing so, it has moved beyond the mission and increased access to transplantation to all patients, regardless of race. Because deceased donor kidney availability has remained inadequate to supply enough kidneys to those in need of renal transplantation, living kidney donation has allowed for more patients to be removed from the waiting list. The opportunities for maximal benefit and flexibility in these approaches are magnified by the inclusion of nondirected *good Samaritan* donors who enter the system with a desire to donate a kidney to someone for purely altruistic purposes. Their inclusion expands the possible paired com-

binations, and in the end, typically results in an additional candidate from the deceased donor kidney waiting list successfully transplanted with a live donor kidney.

Our results firmly make the case that PKDE and antibody reduction therapy can be applied across centers and in large minority populations to increase access for those groups who may benefit the most in receiving a timely successful kidney transplant.

Author Contributions

Study conception and design: Melancon, Rosen-Bronson, Light, Girlanda, Ghasemian, Africa, Johnson

Acquisition of data: Melancon, Cummings, Graham, Rosen-Bronson, Light, Girlanda, Ghasemian, Africa, Johnson

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