ORIGINAL ARTICLE

ABO Blood Type–Incompatible Kidney Transplantation and Access to Organs

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Objective: To determine whether ABO-incompatible (ABOi) kidney transplantation can be performed safely and result in acceptable posttransplantation outcomes.

Design: Prospective study.

Setting: Transplantation center.

Patients: In the 1½ years of a new program, 18 patients with renal failure and an ABOi living kidney donor were included in the study. All donors and recipients were of incompatible blood types and underwent transplantation beginning in June 2008.

Interventions: Patients received immunomodulation (anti-CD20 antibody, intravenous immunoglobulin, and plasmapheresis) until an acceptable isoagglutinin titer was obtained on the date of transplantation. All the kidneys were transplanted heterotopically, and all the patients received induction immunosuppression followed by a combination of prednisone, mycophenolate mofetil, and tacrolimus. Isoagglutinin titers were monitored, and postoperative plasmapheresis was initiated if titers increased.

Main Outcome Measures: Patient and allograft survival; length of stay; 1-, 3-, and 6-month and 1-year renal function; and incidence of rejection.

Results: Patient survival was 100%, with allograft survival of 94.4%. Mean (SD) length of stay was 6.9 (1.9) days. Donor to recipient transplantation was A to O in 11 cases, A2 to B in 1, B to A in 3, B to O in 1, and AB to B in 2. Mean (SD) creatinine levels, a measure of graft function, were 1.2 (0.5) mg/dL at discharge, 1.4 (0.4) mg/dL at 1 month, 1.3 (0.45) mg/dL at 3 months, 1.1 (0.3) mg/dL at 6 months, and 1.2 (0.2) mg/dL at 1 year. One episode of cellular rejection occurred.

Conclusions: These short-term results suggest that with a straightforward regimen, ABOi kidney transplantation is possible, acceptable results and graft function are obtainable, and access to kidney transplantation for those with a blood type–incompatible donor can be expanded.

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In the United States, there are more than 82 000 patients waitlisted for a renal transplant.[1] Routinely, approximately 15% of candidates undergo kidney transplantation in the year after being placed on the waiting list,[2] with a median waiting time to receive a deceased donor renal transplant of 4 years. Waiting list times, particularly for blood group O and B recipients, continue to increase for those waiting to receive a deceased donor kidney transplantation. In some regions, this wait routinely extends beyond 5 years.

One solution to this and the growing number of waiting list candidates is to increase the number of living donor transplantations performed. Potential solutions include (1) transplantation across the ABO blood group barrier, (2) transplantation across a positive crossmatch with desensitization, and (3) paired exchange programs. Although paired exchange programs have assisted some recipients in undergoing transplantation, O recipients remain at a disadvantage because they can receive kidneys from group O donors only, which are in limited supply.

The general approach to overcoming the blood group barrier has been to use a preconditioning regimen. These regimens have included hyperimmune intravenous immunoglobulin (IVIg), plasmapheresis, splenectomy, and, more recently, the use of anti-CD20 therapy. Although regimens have been adopted by a few centers in this country, when a potential living donor kidney candidate is ABO incompatible (ABOi), most transplant programs place the potential recipient on the United Network for Organ Sharing waiting list for a deceased donor kidney, deeming the donor as incompatible. Because waiting times have

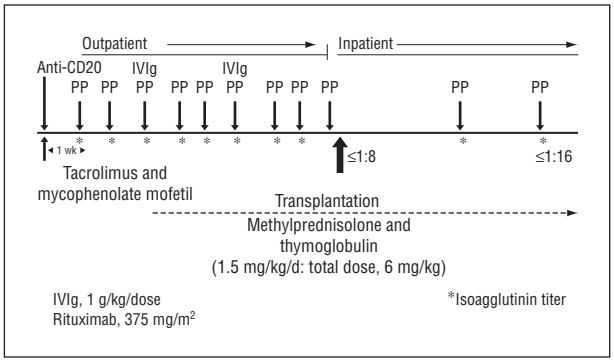

Figure. Protocol for preoperative and postoperative immunomodulation and immunosuppression for ABO-incompatible transplant recipients. IVIg indicates intravenous immunoglobulin; PP, plasmapheresis.

increased, the solution of waiting for a blood group–compatible deceased donor as the only option has become more untenable. Annual mortality for patients on the waiting list is 6% overall and more than 12% for older patients and those diagnosed as having diabetes. The University of California at Los Angeles Transplantation Center developed a protocol for and began an ABOi kidney transplant program in 2008 in part to address the growing waiting list and time to transplantation at the center and in the region. This article describes our single-center experience with ABOi kidney transplantation. The preconditioning regimen and patient and graft outcomes are discussed.

METHODS

PATIENTS AND IMMUNOSUPPRESSION PROTOCOL

Recipients

Between June 1, 2008, and December 31, 2009, 18 consecutive patients underwent ABOi kidney transplantation at the University of California at Los Angeles. After providing informed consent, all the patients were treated with a standardized protocol. A combination of rituximab and 2 g of IVIg were administered 1 month before transplantation. Pretransplantation plasmapheresis was performed 3 times per week to obtain an isoagglutinin IgG titer of 1:8 or less on the day of transplantation. Combination treatment with tacrolimus and mycophenolate mofetil was begun when preoperative plasmapheresis was initiated (**Figure**). Splenectomy was reserved as a posttransplantation salvage maneuver for patients with worsening renal function and a concomitant rise in isoagglutinin titers despite plasmapheresis and IVIg administration.

Donors

Patients donating a kidney underwent a standard workup, including computed tomographic urography, blood work, urinalysis, medical history intake, and physical examination. Cold ischemia time was defined as the interval from the kidney being removed from the donor to removal from cold storage after preparation. Warm ischemia time was defined as the interval between removal of the kidney from cold storage to reperfusion.

IVIg AND PLASMAPHERESIS

Plasmapheresis was performed using therapeutic apheresis (COBE Spectra; CaridianBCT, Lakewood, Colorado). One plasma volume was exchanged with 50%/50% saline/5% albumin. The number of treatments was estimated based on a starting indirect isoagglutinin titer. Pretransplantation isoagglutinin titers were observed during therapy, and adjustments in the pheresis schedule and number of treatments were made accordingly. In all cases, IgG titers were 1:8 or less on the day of transplantation. Posttransplantation pheresis depended on the daily titers. In general, posttransplantation plasmapheresis was performed to keep the titer at 1:8 or less in the first posttransplantation week and at 1:16 or less in the second week. If pheresis was performed, this was followed by the administration of 10 g of IVIg.

TRANSPLANTATION PROCEDURE

Kidneys were procured by laparoscopic nephrectomy and were flushed with crystalloid. Typically, central venous pressure monitoring was used to optimize volume status for recipients. All the grafts were placed in the iliac fossa through an extraperitoneal approach except one in which a left-sided allograft nephrectomy was required before implantation. Extravesicular ureteroneocystostomy was performed with a stent. Intravenous mannitol and furosemide were infused before graft reperfusion in all patients. Additional diuretics and dopamine were administered at the surgeon's discretion.

INTRAOPERATIVE AND POSTOPERATIVE IMMUNOSUPPRESSION

At the time of transplantation, patients received a combination of methylprednisolone (500 mg) and antithymocyte globulin (1.5 mg/kg) induction over 4 days (total dose, 6 mg/kg). Methylprednisolone (250 mg) was administered on postoperative day (POD) 1, 2 mg/kg on POD 2, and 0.5 mg/kg on POD 3. Treatment with tacrolimus was restarted on POD 2 and with prednisone/mycophenolate beginning on POD 4. A tacrolimus level of 10 ng/mL was targeted for the first 3 months, with levels of 6 to 8 ng/mL subsequently. Prednisone tapering was performed to a dose of 5 mg/d during the first 4 weeks (Figure). Recipients of ABOi kidneys from blood group A2 donors underwent preoperative conditioning identical to that of other incompatible blood type recipients.

ANTIBODY TITERS AND CROSSMATCHING

Isohemagglutinin titers were determined by using the standard tube method as described.[3,4] Measurement of titers was performed before each pretransplantation plasmapheresis treatment, daily during the posttransplantation period while hospitalized, and at each outpatient visit during the first posttransplantation month. Patients were seen 3 times weekly for the first 2 weeks, 2 times weekly for weeks 3 and 4, once weekly for the second month, and monthly for 6 months. Crossmatching was performed by flow cytometry and was considered positive when the mean channel shifts were greater than 100 for B cells and greater than 50 for T cells. Anti–HLA class I and II donor-specific antibodies were identified using color-coded multianalyte beads coated with soluble HLA antigen molecules.

DIAGNOSIS AND TREATMENT OF REJECTION

All rejection episodes were biopsy proved. Transplant kidney biopsies were performed for any unexplained rise in serum creatinine level. All the biopsy samples were examined using he-

matoxylin-eosin microscopy and Masson trichrome stain. C4d staining was performed by indirect immunofluorescence. C4d staining and acute cellular rejection were graded according to Banff 2003 criteria.[5] Cellular rejection was treated with a 3-day corticosteroid pulse (5 mg/kg) followed by taper.

STATISTICAL ANALYSIS

Mean, range, and standard deviation were calculated using standard formulae. Data are presented as mean (SD). The study was approved by the institutional review board of the David Geffen School of Medicine, University of California at Los Angeles.

RESULTS

BASELINE CHARACTERISTICS AND DEMOGRAPHICS

The study group comprised 18 adult blood group–incompatible living kidney recipients who underwent transplantation between June 1, 2008, and December 31, 2009. All the patients who were evaluated and expressed a desire to proceed with ABOi kidney transplantation underwent the preconditioning regimen, and none were excluded based on their pretransplantation isoagglutinin titers. All the patients were informed of the kidney paired donation program, and, in general, all were listed for this program.

The mean (SD) age of recipients was 46.0 (13.0) years (age range, 25-65 years), 66.6% were men, and 38.9% were Hispanic and 33.3% were white (**Table 1**). The most common cause of renal failure was hypertension (16.7%). The most common recipient blood type was O (n=12), followed by A (n=3) and B (n=3) (**Table 2**). Starting isoagglutinin IgG titers ranged from 1:1 to 1:1024. The mean starting titer was 1:128. Sensitization to HLA antigen was generally low (mean [SD] [range]: class I, 18.4% [34.6%] [0%-94%]; class II, 9.44% [25.3%] [0%-80%]). Sixty-seven percent of patients had previously had a transfusion, 67% (4 of 6) of the women had had previous pregnancies, and 22% (n=4) had undergone a previous transplantation (heart [n=1], living kidney followed by simultaneous kidney and pancreas [n=1], and kidney alone [n=2]). One patient underwent transplantation across ABOi and positive HLA antigen crossmatch barriers.

The mean (SD) age of kidney transplant donors was 43.4 (13.0) years (age range, 18-64 years), 78% were women, and 44% were white and 33% were Hispanic. Most donors were biologically related (sibling [n=4], parent [n=1], child [n=3], or cousin [n=1]) or spousal (n=6). The remaining were unrelated, with 1 through the paired exchange program. The most common blood type was A (n=12 with A_1=9 and A_2=3) followed by B (n=4) and AB (n=2) (Table 2). Desensitization was initiated in all 18 recipients. The mean (SD) number of plasmapheresis treatments was 9 (3) before transplantation and 6 (5) after transplantation (**Table 3**).

The mean (SD) cold ischemia time was 2:11 (1:46) hours (range, 1:24-8:48 hours) and anastomosis time was 47 (5) minutes (range, 36-54 minutes). Mean (SD) number of HLA antigen matches was 2.2 (1.6). Posttransplantation mean (SD) length of stay was 6.9 (1.9) days, with a discharge creatinine level of 1.2 (0.5) mg/dL (to convert to micromoles per liter, multiply by 88.4). Six patients were readmitted once after transplantation: 2 for

Table 1. Baseline Characteristics of 18 ABO-Incompatible Renal Transplant Recipients

Characteristic	Value
Age, y	
Mean (SD)	46.0 (13.0)
Range	25-65
Sex, %	
Male	66.6
Female	33.3
Race, %	
White	33.3
Black	16.7
Hispanic	38.9
Asian	11.1
Cause of renal failure, %	
Hypertension	16.7
Polycystic kidney disease	11.1
IgA nephropathy	11.1
CNI-induced nephrotoxicity	11.1
FSGS	11.1
Unknown	11.1
SLE	5.6
C1q nephropathy	5.6
Chronic glomerulonephritis	5.6
Membranous glomerulonephritis	5.6
Obstructive uropathy	5.6
Dialysis, %	
Dialysis	77.8
Predialysis	22.2
Sensitization, %	
Nonsensitized	66.7
Peak PRA >30%	22.2
Previous transplantation	22.2
Previous transfusion	66.7
Starting isoagglutinin, IgG, titer	
Mean	1:128
Range	1:1-1:1024
Blood type	
A	3
B	3
O	12

Abbreviations: CNI, calcineurin inhibitor; FSGS, focal segmental glomerulosclerosis; PRA, panel reactive antibody; SLE, systemic lupus erythematosus.

Table 2. Donor and Recipient Blood Type Combinations and Number of Patients

Blood Type		
Donor	Recipient	Patients, No.
A1	O	9
A2	O	2
B	O	1
AB	O	0
AB	A	0
AB	B	2
A1	B	0
A2	B	1
B	A	3

Table 3. Range of Plasmapheresis Treatments Before and After Transplantation

Starting Isoagglutinin IgG Titer	Treatments, No.	
	Pretransplantation	Posttransplantation
Negative	0	1
16	4-5	0-1
32	5-6	0-1
64	4-7	1-8
128	5-8	2-6
256	8-13	5-11
512	14	6
1024	18	12

an elevated creatinine level (1 of whom was diagnosed as having rejection), 1 after a rise in the IgG isoagglutinin titer for multiple days of inpatient plasmapheresis, 1 for severe nausea after plasmapheresis, 1 for intractable diarrhea, and 1 for treatment of hyperglycemia.

GRAFT AND PATIENT SURVIVAL, INCIDENCE OF REJECTION, AND OTHER COMPLICATIONS

No deaths occurred in the series. Patient survival to date is 100%. Graft survival was 100% at 1 week and 94.4% at 1, 3, 6, and 12 months. One patient developed BK viuria and viremia 2 months after transplantation. Resolution of BK viremia occurred 2 months after reduction in maintenance immunosuppression, and the creatinine level has remained at 0.9 mg/dL.

All the grafts functioned properly at the time of transplantation, and urine was either made in the operating room after reperfusion or shortly afterward in the recovery room. The mean (SD) creatinine level at discharge was 1.2 (0.5) mg/dL, at 1 month was 1.4 (0.4) mg/dL, at 6 months was 1.1 (0.3) mg/dL, and at 1 year was 1.2 (0.2) mg/dL. One graft loss was due to acute antibody-mediated rejection (AMR) in a negative crossmatch patient with a moderately high pretransplantation IgG titer (1:256). Pretransplantation panel reactive antibody classes I and II were 0% and 13%, respectively. The patient's IgG titer was 1:8 on POD 1 and rose to 1:16 on POD 2, prompting plasmapheresis. The IgG titer returned to 1:8 on POD 3 and remained at 1:8 on POD 4. On PODs 5 and 6, plasmapheresis was performed for IgG titers of 1:16 and 1:32, respectively. The patient otherwise had initial good graft function, with the creatinine level decreasing to 1.9 mg/dL by POD 6. On POD 7, however, anti-A1 IgM titers increased from 1:8 to 1:32, whereas IgG titers remained unchanged. The creatinine level remained stable at 1.9 mg/dL. In the next 24 hours (POD 8), the patient's creatinine level increased to 4.4 mg/dL concomitant with a sharp rise in IgM and IgG titers despite plasmapheresis the preceding day (IgM titer, 1:32→1:128; and IgG titer, 1:32→1:256). Clinically, the patient became oliguric. Doppler ultrasound demonstrated no vascular flow in the parenchyma, prompting exploratory laparotomy. Intraoperatively, the kidney was found to be enlarged and cyanotic, without vascular signals. Allograft nephrectomy was performed, and the patient was reinitiated on hemodialysis. Pathologic findings demonstrated severe AMR. No other patients required dialysis at any time during their in-hospital or outpatient course. No other cases of AMR occurred.

There was 1 case of a moderate rise in IgG isoagglutinin titer that prompted emergent splenectomy in conjunction with plasmapheresis and IVIg therapy. The patient had immediate function, with the creatinine level decreasing to 1.9 mg/dL by POD 4; the IgG titer was 1:8. On POD 5, the titer increased to 1:16, prompting plasmapheresis. On POD 6, the titer increased to 1:64, with a concomitant rise in the serum creatinine level to 2.2 mg/dL. Splenectomy was performed owing to the concern for the imminent development of acute AMR. The patient's titer declined to 1:32 the subsequent day, and plasmapheresis was continued for the next 5 days. The patient's creatinine level declined to 1.7 mg/dL, and his titer has since remained at 1:8 or less (16 months postoperatively at the time of this writing). His allograft function remains stable, without evidence of AMR. No other cases of rising IgG titer associated with elevation in creatinine level were noted.

In 1 case, an allograft biopsy specimen demonstrated borderline inflammation and tubulitis suspicious for acute cellular rejection, tubulointerstitial type. The patient responded to a pulse of methylprednisolone, with subsequent corticosteroid taper. No patients required a second operation, and there were no abscesses, urine leaks, cytomegalovirus infections, wound infections, or urinary tract infections in the immediate posttransplantation period in these patients.

COMMENT

Given the general scarcity of deceased donor organs for transplantation, efforts should continue to stress improving the availability of organs from living donors. Living kidney donation has advantages over deceased donation, including improved initial graft function, increased long-term graft survival, and the potential for transplantation to be performed before the initiation of dialysis.[4] Although the number of patients waiting for a kidney transplant continues to increase and the number of deceased organ donors remains relatively flat, living donor kidney transplantation continues to be considered to make up for this ever-growing disparity. In addition, as those previous kidney transplant recipients reach the natural end of life of their graft, the transplant community will face an increasing number of recipients waiting to undergo transplantation again. Although some patients have motivated prospective donors, many are found to be ABOi or to possess HLA antigen to which the recipient is sensitized. Indeed, ABOi has been suggested to be one of the most significant barriers to expansion of live kidney donation.[6] Our single-center database revealed that, in 2007, ABOi- and HLA antigen–crossmatch positive donor-recipient pairs represented the largest group among inactive donors and composed almost 19% of inactive donor candidates (89 of 475). Fifty-seven and 32 donors were deemed incompatible with their respective recipients due to ABOi and positive crossmatches, respectively (G.S.L., unpublished data, 2009).

Early attempts to cross such barriers led to hyperacute rejection and graft loss,[7,8] but in the 1980s[9-11] and early 1990s,[12] successful ABOi kidney transplantation was occasionally reported but generally unaccepted. Although some of these initial successes involved an A2 donor[13] (less immunogenic), later studies[14] demonstrated success with non-A2 kidneys. Although these protocols may have been viewed as cumbersome because of their requirement for local irradiation and splenectomy,[15] newer protocols do not require splenectomy or irradiation, and transplantation can generally be performed if the recipient's isoagglutinin IgG titer does not exceed 1:8 on the day of transplantation. After the first 2 weeks after transplantation, isoagglutinin titers may be allowed to increase without treatment if no renal dysfunction occurs. In fact, in 10% of our patients, the titer elevated without evidence of renal dysfunction, suggesting that immunologic accommodation is occurring.

We believe that there is a lifelong advantage to avoiding splenectomy unless needed for rescue therapy. It should be reserved for patients who are recalcitrant to postoperative plasmapheresis, with rising isoagglutinin titers associated with renal dysfunction, or for those who develop severe refractory AMR. However, with the urgency and threat to graft function and the time constraint in making a diagnosis, it is not always clear as to when a splenectomy should be performed. In our limited experience, we advocate splenectomy early after transplantation if there is a substantial rise in titers refractory to plasmapheresis, AMR is a likely diagnosis, and renal dysfunction is apparent.

Baseline pretransplantation isoagglutinin IgG titers seem to be the best predictor of the need for posttransplantation plasmapheresis to keep isoagglutinin titers low during the period of accommodation. In general, patients with starting titers of 1:32 or less were likely to require few, if any, posttransplantation plasmapheresis treatments. Conversely, patients with higher baseline titers, such as 1:512 and 1:1024, may require 6 to 12 pheresis treatments after transplantation to keep isoagglutinin titers low in the early posttransplantation period.

Early studies suggest that patients with elevated isoagglutinin IgG titers and rebound antibody production after antibody removal may be at higher immunologic risk. However, excellent short- and long-term patient and graft survival rates have been achieved in patients with high isoagglutinin IgG titers before plasmapheresis or immunoadsorption and maintained on immunosuppression that includes tacrolimus and mycophenolate. In a study of 167 recipients who underwent ABOi transplantation, Shimmura et al[16] reported no correlation between ABO isoagglutinin titers and graft survival in patients who received tacrolimus and mycophenolate immunosuppression. In addition, Montgomery et al[17] reported no increase in AMR in patients with high initial isohemagglutinin titers in their series of 60 patients receiving tacrolimus and mycophenolate as part of their immunosuppression.

This experience with a simplified preconditioning regimen demonstrates that excellent 1-year patient and graft survival rates can be obtained with an ABOi kidney transplant. Early experiences using sophisticated pretransplantation preconditioning regimens that included splenectomy have demonstrated similar short-term patient and graft survival rates.[18] For example, in a series of 18 ABOi transplant recipients, 10 of whom underwent splenectomy at the time of transplantation, 1-year patient and graft survival of 96% and 89%, respectively, were achieved. In another study,[19] 20 ABOi recipients with more than 12 months of follow-up were compared with ABO-compatible living donor recipients. There was no significant difference in patient or graft survival or in the incidence of acute rejection. In adult kidney recipients, the mean glomerular filtration rate was equivalent at all time points. The results of these studies and the work of others suggest that ABOi kidney transplantation is an excellent alternative for those who have a motivated but ABOi living donor while avoiding a prolonged wait on a deceased donor waiting list.

Although follow-up was short for this study, others using alternative protocols have demonstrated 5-year graft survival of 89%[17] and 8-year graft survival of 73%.[14] These other groups, at times, have used techniques or agents, including splenectomy, local irradiation of the graft, and deoxyspergaulin,[20] along with other components of an intensive immunosuppressive regimen, whereas the simplified protocol that we developed does not. The present study requires longer follow-up to determine whether similar long-term graft survival can be achieved with this protocol in this patient group.

The keys to a successful ABOi kidney transplantation program under this protocol are having a planned strategy for pretransplantation desensitization and careful follow-up in the immediate posttransplantation period. Careful planning involves choosing appropriate recipients, ensuring appropriate intraoperative management of blood products, and ensuring antibody removal postoperatively if needed. Although in aggregate successful ABOi kidney transplantation is possible, the failure of an individual case accentuates that uncertainty remains about the clinical significance of small fluctuations in antibody titers and creatinine levels in the early postoperative period. When uncertainty arises, diagnostic studies, such as ultrasonography and early allograft biopsy, and intensive plasmapheresis should be performed at the discretion of the physician. However, we believe that with diligence, experience, and intense clinical acumen, ABOi kidney transplantation can be successful in many patients and can increase access to organs for those with a living kidney donor.

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