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Cancer Risk Following ABO Incompatible Living Donor Kidney Transplantation (1)

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Abstract

Background—Recipients of ABO incompatible (ABOi) living donor kidney transplants often undergo more intense immunosuppression than their ABO compatible (ABOc) counterparts. It is unknown if this difference leads to higher cancer risk after transplantation. Single-center studies are too small, and lack adequate duration of follow-up, to answer this question.

Methods—We identified 318 ABOi recipients in the Cancer Transplant Match Study, a national linkage between the Scientific Registry of Transplant Recipients and population-based U.S. cancer registries. Seven cancers (non-Hodgkin lymphoma, Merkel cell carcinoma, gastric adenocarcinoma, hepatocellular carcinoma, thyroid cancer, pancreatic cancer, and testicular cancer) were identified among ABOi recipients. We then matched ABOi recipients to ABOc controls by age, gender, race, HLA mismatch, retransplantation, and transplant year.

Results—There was no demonstrable association between ABOi and cancer in unadjusted incidence rate ratio (IRR 0.83, 95% CI 0.33–1.71, p=0.3) or matched control analysis (IRR 0.99, 95% CI 0.38–2.23, p=0.5).

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Conclusion—To the extent that could be determined in this registry study, current desensitization protocols are not associated with increased risk of cancer after transplantation.

Keywords

incompatible transplantation; cancer; living donor kidney transplantation

Introduction

ABO incompatible (ABOi) living donor kidney transplantation is becoming increasingly common, largely as a response to continuing shortage of kidney donors. Since 2006, ABOi transplants comprise 1.5% of all living donor transplants in the United States [1]. In most reports, ABOi recipients have similar patient and graft survival to their ABO compatible (ABOc) counterparts [1–4]. In order to achieve these results, most ABOi kidney recipients undergo more intense immunomodulatory protocols that include plasmapheresis, intravenous immune globulin, anti-CD20 treatment, and/or splenectomy [5, 6].

In general, the cancer risk for organ recipients is increased, due largely to immunosuppression [7]. This increased risk is particularly pronounced among infection-related cancers and ranges from 1.5-fold increased risk for stomach cancer to 61-fold increased risk for Kaposi sarcoma. Individual steps of ABOi protocols, including splenectomy and other forms of B-cell modulation, are associated with mildly increased cancer risk in other contexts [8–10]. It is possible that these protocols might further increase the risk of cancer after transplantation, although this has never been studied.

As ABOi transplantation becomes more common and survival improves, it is necessary to evaluate the risks of long-term complications such as cancer in order to tailor patient selection, consent, screening, and prevention appropriately. Our objective was to compare cancer risk in equivalent ABOi versus ABOc living donor kidney transplant recipients using the Transplant Cancer Match (TCM) Study, a linkage between the Scientific Registry of Transplant Recipients (SRTR) and U.S. population-based cancer registries [7]. The TCM offers the first opportunity to study high quality cancer follow-up data in a large, national cohort of ABOi recipients.

Results

Comparing 318 living donor ABOi kidney recipients with 37,643 ABOc recipients during the study period, age at transplantation, gender, race, percentage of retransplants and zero HLA mismatch status were similar. However, a higher percentage of ABOi recipients were African-American (19.3% vs. 14.0%, p=0.02) and had received a retransplant (11.0% vs. 8.0%, p=0.03) (Table 1).

As expected, ABOi transplantation was skewed towards more recent years, with 55.4% of ABOi transplants performed between 2004 and 2008. The total time at risk for ABOi recipients was 990.7 person years (median 2.00 years). An A donor to 0 recipient was the most common type of ABOi (27.0%) (Table 2).

Among ABOi recipients, there were seven cancers identified with one case each of non-Hodgkin lymphoma (NHL), Merkel cell carcinoma (MCC), gastric adenocarcinoma, hepatocellular carcinoma, papillary thyroid cancer, pancreatic cancer, and testicular germinoma. Four of these cancers were infection-related (NHL, Merkel cell carcinoma, gastric adenocarcinoma, hepatocellular carcinoma). The time to cancer diagnosis ranged from 0.9 to 9.2 years (median 3.6 years). ABOi recipients had no demonstrable difference in

overall cancer risk compared to ABOc recipients in unadjusted (IRR 0.83, 95% CI 0.33–1.71, p=0.3) or matched (IRR 0.99, 95% CI 0.38–2.23) analysis (Table 3).

The NHL case diagnosed among the ABOi recipients was a nodal Burkitt lymphoma. The time to diagnosis was 5.9 years. ABOi recipients had no demonstrable difference in NHL risk compared to ABOc recipients in unadjusted (IRR 0.86, 95% CI 0.02–4.85, p=0.5) or matched (IRR 1.02, 95% CI 0.02–8.38, p=0.5) analysis.

Discussion

In this first, limited exploration of cancer after ABOi transplantation, using a national linkage of transplant registry to cancer registry data, we did not detect an elevated post-transplant cancer risk associated with ABO incompatibility.

Only one ABOi recipient was diagnosed with NHL, typically the most common malignancy after transplantation (except for basal and squamous cell skin cancers). There was not a demonstrable difference between the incidence rate of NHL in ABOi and ABOc recipients. The single case of NHL was diagnosed at 5.9 years, consistent with the late peak of NHL risk after transplantation [11]. While in general, late NHL is less likely to be Epstein-Barr virus associated and more likely to be extra-nodal [12], the NHL diagnosed among the ABOi recipients was Burkitt lymphoma and nodal. Burkitt lymphoma risk is increased in association with immunosuppression due to HIV infection or transplantation, and possibly related to EBV infection [13–15]. Somewhat surprisingly, there were no diagnoses of early NHL (within 2 years after transplant) among ABOi recipients. Anti-CD20 antibodies are given as part of incompatible desensitization protocols at certain centers and are also used in the treatment of NHL [16, 17]. Anti-CD20 antibodies deplete B-cells that may contribute to development of NHL. The peak period of B-cell immunomodulation with anti-CD20 is during the peak of early risk for NHL [18]. It is possible that the anti-CD20 antibodies could decrease risk of NHL, particularly during this early period. More targeted research into the associations of this immunomodulation and NHL risk should be performed.

Of interest, Merkel cell carcinoma, a rare neuroendocrine tumor of the skin associated with immunosuppressed states and thought to be caused by Merkel cell polyoma virus [19], was diagnosed among the ABOi cohort. Merkel cell carcinoma risk is elevated among transplant recipients [20, 21]. Increased risk has also been found in HIV-positive patients [22] and associated with chronic lymphocytic leukemia (CLL) [23, 24]. Prominent immune dysfunctions in CLL include B-cell dysfunction and hypogammaglobulinemia [25–27]. B-cell deficits are also induced in ABOi recipients as part of desensitization protocols and may offer a mechanistic explanation for the development of this rare cancer.

Strengths of our study include the use of a national cohort of living donor kidney recipients and accurate cancer ascertainment independent of transplant center follow-up and reporting. Using cancer registry linkage allowed for the greatest and most accurate follow-up time possible for each of the ABOi kidney transplant recipients captured; however, because the practice of ABOi transplantation is a relatively recent one, our median follow-up time could only be 2 years. It is possible that differences in cancer risk will become apparent when increasing numbers of ABOi recipients are followed for longer periods of time. While our cohort is the largest to date in answering this question, it is nonetheless too small to allow additional interesting analyses such as stratification by blood type, other recipient characteristics, or cancer types. Limitations also include lack of antibody titer in SRTR data, minimal information about desensitization protocols, and likely heterogeneity in practice patterns throughout the country. Another limitation is the lack of information on the incidence of non-melanoma skin cancer, the most common cancer after transplantation.

Expansion of ABOi kidney transplantation offers hope for increasing available kidney donors and access to transplantation. As outcomes after ABOi transplantation improve, it will be necessary to closely study the possible long-term risks associated with this procedure. Using the largest, albeit not large, national cohort to date, we were unable to demonstrate differences in cancer risk associated with ABOi compared with ABOc kidney transplantation. Further efforts should be made to capture accurate information on additional incompatible transplants and to track the long-term outcomes in this unique cohort of recipients.

Methods

Eligible living kidney recipients were identified in the TCM Study, a linkage of data from SRTR (1987–2008) with 14 population-based cancer registries throughout the United States (http://transplantmatch.cancer.gov/). The SRTR includes data on all U.S. solid organ transplants. Participating cancer registries, which together cover approximately 43% of the U.S. transplant population, ascertained the occurrence of malignancies (other than basal cell and squamous cell skin cancer) based on mandatory reporting from hospitals, medical providers, and pathology laboratories. Following linkage with the SRTR, investigators retained only anonymized data from the cancer registries. The study was approved by human subjects committees at the National Cancer Institute and, as required, at participating cancer registries.

Eligible recipients were those that received an ABOi living donor kidney transplant during a time period with available data on cancer from participating registries. We used the linked cancer registry data to identify first incident cancer cases following transplantation. Cancers were classified and recorded by cancer registries using topography and morphology codes. Follow-up started at transplantation and ended at death, graft failure, retransplantation, loss of follow-up, or end of cancer registry coverage.

Unadjusted incidence rate ratios and exact 95% confidence intervals for ABOi vs. ABO compatible (ABOc) living donor kidney recipients were calculated for all cancers and separately for non-Hodgkin lymphoma (NHL). In addition, a matched control cohort was created by matching ABOc living kidney recipients 5-to-1 with ABOi recipients. Matches were drawn from 37,643 possible ABOc controls available in SRTR data. Using iterative expanding radius matching as previously described [28, 29], each control was matched on age at transplantation, gender, race, zero HLA mismatch status, retransplantation, and year of transplant. Based on an incidence rate of 0.0085 per year in the ABOc cohort, we had 80% power to detect a 3-fold increase in cancer incidence based on our cohort of 318 ABOi patients.

All analyses were performed using Stata 12.0/MP for Linux (StataCorp, College Station, TX, www.stata.com).

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Abbreviations

AAMR	acute antibody mediated rejection
ABOc	ABO compatible
ABOi	ABO incompatible
IVIG	intravenous immune globulin
SRTR	Scientific Registry of Transplant Recipients
TCM	Transplant Cancer Match

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Table 1

Demographics of living donor kidney recipients in the Transplant Cancer Match Study by ABO compatibility status

	ABOi	ABOc (entire cohort)	P-Value	ABOc (matched controls)[a]
	N=318	N=37,643		N=1,590
Age			0.4	
0–35	107 (33.7)	13,669 (36.3)		550 (34.6)
36–50	99 (31.1)	12,392 (32.9)		467 (29.2)
51–60	70 (22.0)	7,357 (19.5)		374 (23.5)
>60	42 (13.2)	4,225 (11.2)		199 (12.5)
Gender			0.5	
Male	181 (56.9)	22,067 (58.6)		905 (56.9)
Female	137 (43.1)	15,576 (41.4)		685 (43.1)
Race[b]			0.02	
White	190 (60.1)	23,208 (62.1)		954 (60.0)
African-American	61 (19.0)	5,238 (14.0)		307 (19.3)
Hispanic/Other	65 (20.6)	8,941 (23.9)		329 (20.7)
Zero HLA Mismatch[b]			0.6	
No	274 (88.7)	32,377 (87.6)		1,410 (88.7)
Yes	35 (11.3)	4,575 (12.4)		180 (11.3)
Retransplantation			0.03	
No	282 (88.7)	34,633 (92.0)		1,410 (88.7)
Yes	36 (11.3)	3,010 (8.00)		180 (11.3)

ABOi = ABO incompatible; ABOc = ABO compatible. Cells show N (%).

a) Matched 5 to 1 on age at transplantation (within five years), gender, race, zero HLA mismatch status, retransplantation, and year of transplant (within ten years) to ABOi recipients.

b) Recipients do not sum to total because of missing data. P-values represent the comparison between ABOi patients and the entire ABOc cohort, using a chi-squared test.

Table 2

Types of living donor ABO incompatible transplants in the Transplant Cancer Match Study

Recipient Blood Type	Donor Blood Type						
	A	**A1**	**A1B**	**A2**	**A2B**	**AB**	**B**
O	87 (27.4)	17 (5.4)	0 (0)	60 (18.9)	0 (0)	4 (1.3)	49 (15.4)
A	-	-	1 (0.3)	-	1(0.3)	5 (1.6)	35 (11.0)
B	29 (9.1)	2 (0.6)	0 (0)	9 (2.8)	9 (2.8)	10 (3.1)	-

Cells show N (%), where percentages are of total ABOi (N=318).

Table 3

Cancer risk after living donor kidney transplantation, comparing ABO incompatible recipients with ABO compatible recipients and matched ABO compatible controls

	ABOi	ABOc (entire cohort)	ABOc (matched controls)[a]
All Cancer			
Rate[b]	7.1	8.5	7.1
IRR (95% CI) vs. entire cohort	0.86 (0.02–4.85)	Reference	
IRR (95% CI) vs. matched controls	0.99 (0.38–2.23)		Reference
NHL			
Rate[b]	1.0	1.2	1.0
IRR (95% CI) vs. entire cohort	0.76 (0.02–4.29)	Reference	
IRR (95% CI) vs. matched controls	1.02 (0.02–8.38)		Reference

ABOi = ABO incompatible; ABOc = ABO compatible; IRR = incidence rate ratio; CI = confidence interval; NHL = non-Hodgkin lymphoma.

[a] Matched 5 to 1 on age at transplantation, gender, race, zero HLA mismatch status, retransplantation, and year of transplant to ABOi recipients.

[b] Per 1,000 person-years