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Donor Age Is Associated With Chronic Allograft Vasculopathy After Adult Heart Transplantation: Implications for Donor Allocation

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Abstract

Background—Chronic allograft vasculopathy (CAV) is a major cause of long-term complications and mortality after heart transplantation. Although recipient factors have been implicated, little is known of the role of donor factors in CAV development. We sought to identify donor factors associated with development of CAV after heart transplantation.

Methods—We reviewed the United Network for Organ Sharing heart transplant database from August 1987 to May 2008. Univariate and multivariate analyses were performed to assess the association between donor variables and the onset of CAV for adult recipients. Donor age was matched to recipient age and analyzed with respect to development of CAV.

Results—Of the 39,704 recipients, a total of 11,714 (29.5%) experienced CAV. Multivariate analysis demonstrated seven donor factors as independent predictors of CAV: age, ethnicity, sex, weight, history of diabetes, hypertension, and tobacco use. When matching young donors (0 to 19.9 years) and old donors (≥ 50 years) to each recipient age group, older donors (≥ 50 years) conferred a higher risk of developing CAV. Further modeling demonstrated that for each recipient group, older donor age (≥ 50 years) conferred a higher risk of CAV development compared with younger donor age (0 to 19.9 years; $p < 0.0001$).

Conclusions—Donor factors including sex, hypertension, diabetes, and tobacco use are independently associated with recipient CAV. Older donor age confers a greater risk of CAV development regardless of the age of the recipient. A heightened awareness for the development of CAV is warranted when using older donors in adult cardiac transplantation, in particular with recipients 40 years of age or older.

Shortage of donor heart allografts remains the major limitation in cardiac transplantation; thus, the optimal allocation of donor hearts is paramount [1,2]. Although early survival after heart transplantation is limited by acute rejection, the most common cause of late (>5 years) deaths is chronic allograft vasculopathy (CAV) and graft failure [3]. Chronic allograft vasculopathy is a hastened form of coronary artery disease in the transplanted heart. This disease process is characterized by concentric fibrous intimal hyperplasia in coronary vessels and is difficult to diagnose. Although angiography remains the gold standard for detecting focal plaques, it lacks the sensitivity to detect subclinical CAV. Although more

sensitive, intravenous ultrasonography lacks the ability to interrogate the entire coronary vasculature [4].

Recent reports have identified that both recipient and donor variables are associated with the development of CAV. Recipient factors include male sex, pretransplant ischemic disease, older age, and black race; donor factors include history of hypertension, older age, and male sex [5–7]. Although these studies have allowed an understanding of risk factors associated with CAV, they do not provide a strategy to potentially reduce the incidence of CAV.

There remains limited information on which clinical donor variables contribute to the development of CAV and which factors are associated with long-term CAV-free survival. In this report, we used the United Network of Organ Sharing (UNOS) database to identify donor factors related to the long-term development of CAV. Moreover, because older donor age has been associated with a higher risk of CAV, we further studied the impact of donor age by examining the relationship between the onset of CAV and donor and recipient age.

Patients and Methods

Data Source

The UNOS cardiac transplant database, a prospectively collected database of all cardiac transplantations in the United States, from August 1987 to May 2008 was used. The database includes transplant and follow-up information from the UNOS Standard Transplant Analysis and Research files with identifying patient and center information excluded. All cardiac transplant recipients had yearly follow-ups.

Patient Population

The UNOS database was queried for all cardiac transplant recipients. The resulting cohort of patients was stratified based on their CAV status. The UNOS organization does not provide a standard method by which to detect CAV in recipients. Thus, the diagnosis of CAV is dependent on the individual transplant centers' screening algorithm and is self-reported. Recipients with missing or unknown status regarding CAV or those who were younger than 18 years of age were excluded.

Variables

Those donor factors included within the study were chosen based on perceived clinical relevance and previous peer-reviewed studies. These included sex, age, race, cause of death, creatinine level, presence of active blood infection, cytomegalovirus match status, and history of hypertension, diabetes, tobacco use, alcohol, and intravenous drug abuse.

The following additional variables were created to allow for comparisons between donor and recipient populations: elevated donor weight (≥ 75 th percentile) and elevated creatinine (≥ 75 th percentile). For the purpose of aged-matched analysis, the donor and recipient populations were grouped by age: stratified recipient age (18–29.9, 30–39.9, 40–49.9, and ≥ 50 years) and stratified donor age (0–19.9, 20–29.9, 30–39.9, 40–49.9, and ≥ 50 years).

Statistical Analysis

Demographics and preoperative factors were evaluated by univariate analysis for recipients and their donors with respect to CAV development. These survivors were further stratified into patients with and without CAV. Categorical variables were compared using Fisher's exact or χ^2 tests as appropriate. Continuous variables were assessed using the Mann-Whitney *U* test.

A multivariate model was created to identify independent donor variables associated with development of CAV using all adult patients with known CAV status. All selected covariates, including variables not significant on univariate analysis, were entered into the logistic regression model. Those patients with incomplete information for a specific variable were classified as unknown for that variable and included within the multivariate model to reduce the selection bias of the study. Significant donor factors from univariate analysis as well as those identified in the literature were entered into a logistic regression model including age, sex, cytomegalovirus status, ethnicity, weight, creatinine, history of diabetes, history of hypertension, history of alcohol use, history of tobacco use, history of intravenous drug abuse, and death owing to trauma. A c statistic was calculated to measure the discriminative power of the logistic equation.

Kaplan-Meier analysis and log-rank tests were used to determine the influence of donor and recipient age on survival with and without CAV. The resultant age-matched Kaplan-Meier curves generated relative risks for the development of CAV with respect to donor and recipient age. Risk ratios were produced by comparing the relative risks of transplanting a recipient with an old (≥ 50 years) or young (10 to 19.9 years) donor heart.

All statistical analysis was performed with SAS version 9.1.3 (SAS Institute, Cary, NC). A probability value of less than 0.05 was considered significant. All means are represented as mean $\pm$ standard deviation.

Results

Demographics and Recipient Variables

The UNOS database yielded 53,039 cardiac transplantations from August 1987 through May 2008. Of these, 7,803 (14.7%) had missing or unknown CAV status and were therefore excluded from the study cohort. Moreover, recipients younger than 18 years of age (5,532; 12.2%) were excluded. The final study population comprised 39,704 adult cardiac transplant recipients with known CAV status. This cohort included patients who were more likely male, older than 40 years of age, and white. Of these recipients, 27,990 (70.5%) patients were free from CAV whereas 11,714 (29.5%) patients experienced CAV (Table 1). The prevalence of CAV did not decrease during the study period, with $23.0\% \pm 3.4\%$ of recipients experiencing CAV 5 years after transplant from 1987 through 2003.

Donor Variables

Donors of recipients in the study cohort were commonly male, younger than 40 years of age, and white, and they died secondary to trauma. Less common donor factors included history of tobacco use, history of alcohol, creatinine values of at least 1.5 mg/dL, history of hypertension, active blood infection, history of diabetes, and history of intravenous drug abuse (Table 2).

Univariate Analysis of Donor and Recipient Factors Related to Chronic Allograft Vasculopathy

Several recipient variables were associated with CAV development, including recipient age ($p < 0.0001$). Multiple donor variables were associated with recipient CAV, including male donors. Other donor variables associated with CAV included older age, donor ethnicity, death as a result of trauma or stroke, and creatinine values of at least 1.5 mg/dL (Table 3). Importantly, the presence of a cytomegalovirus-matched organ, heavier donor weight, donor history of diabetes, and donor history of intravenous drug abuse were not associated with CAV development in the recipient.

Multivariate Analysis of Risk Factors Related to Chronic Allograft Vasculopathy

A multivariate model (c statistic = 0.68) was created using variables identified a priori from the literature as important factors associated with CAV development. Among covariates, seven donor factors were identified as independent predictors of late CAV (Table 4). Of relevance to the present study, donor age had a highly significant association with development of CAV. Logistic regression demonstrated that increasing donor age was associated with greater risk of CAV. Other donor variables independently associated with CAV included donor tobacco use, elevated donor weight, and donor history of diabetes or hypertension. However, both female sex and donor race other than white conferred a lower risk for the development of CAV.

Age-Matched Analysis of Recipient and Donor Populations

Given the highly significant association of donor age with CAV, we hypothesized that the relationship between older donor age and CAV is dependent on recipient age. Using all 39,704 adult heart transplant recipients, donors were matched to their recipients stratified by age and analyzed with respect to the development of CAV.

By Kaplan-Meier and relative risk analyses, young recipients (18 to 29.9 years) had a higher risk that trended toward experiencing CAV during their lifetime when they received a heart from an old donor (≥ 50 years) compared with a young donor (0 to 19.9 years; risk ratio, 1.22; 95% confidence interval, 0.91 to 1.65; $p > 0.05$). Furthermore, old recipients (≥ 50 years) had a higher risk than young recipients of experiencing CAV when receiving a heart from an older donor compared with a young donor (risk ratio, 1.34; 95% confidence interval, 1.25 to 1.43; $p < 0.05$; Fig 1). These findings suggest that older donor age is associated with chronic rejection and CAV development and that old donor age appears to have a greater negative effect than younger donors regardless of recipient age.

Further investigation was performed of donors 0 to 19.9 years and donors at least 50 years age-matched to all recipient populations (18 to 29.9 years, 30 to 39.9 years, 40 to 49.9 years, and ≥ 50 years). The risk ratios for developing CAV and probability values for each permutation were calculated (Table 5). For each recipient age group, older donor age (≥ 50 years) conferred a higher risk of CAV development compared with younger donors (0 to 19.9 years). This finding is especially pronounced in recipients at least 40 years old. Additional analysis demonstrated that for each permutation of donors and recipients, recipients of older donor hearts had a higher risk of CAV development (Fig 2). These results suggest that receiving an older donor heart may impart a baseline risk for the development of CAV.

Comment

Chronic allograft vasculopathy, an accelerated form of coronary artery disease in the transplanted heart, is a major factor that limits long-term survival after heart transplantation [4,8]. In the present study using the UNOS database, a number of donor factors were found to be independently associated with the development of CAV. We further demonstrated a significant relationship between donor and recipient age, suggesting that older donor hearts confer an increased risk of CAV compared with young donor hearts regardless of recipient age and that the risk is significant for those recipients older than the age of 40 years.

Although early survival after heart transplantation is limited by acute rejection, annual reports of the Registry of the International Society for Heart and Lung Transplantation (ISHLT) have suggested that CAV combined with late graft failure (likely because of allograft vasculopathy) accounted for 33% of deaths for those recipients who survived 5 years after transplant, followed in frequency by malignancies and non-cytomegalovirus

infections [3]. Additional reports from the ISHLT have shown that by 10 years after transplantation, approximately 50% of recipients had angiographic evidence of CAV [7]. However, we believe that by limiting our evaluation of CAV to those recipients who survived 5 or even 10 years, we neglect a significant portion of the cardiac recipient population that may have experienced the effects of CAV.

Previous reports have studied both allodependent [9–13] and alloindependent [14–17] factors associated with graft failure and overall survival after heart transplantation. Recipient factors associated with increased risk have been identified in smaller studies and databases and include male sex, ischemic cardiomyopathy, older age, and black race. Donor factors associated with increased risk include history of hypertension, older age, and male sex [5–7,9]. In the present study, we focused primarily on alloindependent factors, in particular age and its relation to CAV development, to provide insight on the effects of donor variables on the late development of CAV. A recent study from the ISHLT reported that elevated donor age was an independent predictor of CAV development. In this study, donor age was treated as a continuous variable and therefore it remained unclear as to whether increased age serves as occult pretransplant coronary artery disease or an age-related phenomenon [7]. Our study supports the findings in the report from the ISHLT identifying increasing donor age as a predictor of the development of CAV. However, our study attempts to provide a strategy by which to potentially reduce the incidence of CAV by carefully analyzing the interaction between donor and recipient age with respect to the development of CAV.

There are many speculations to explain our findings. The most likely explanation is that older donor hearts have subclinical coronary disease that result in the earlier development of clinically significant coronary disease. Other potential reasons may include the resistance of young donors to coronary artery disease, or alloimmunologic factors in the older donor heart that make it more prone to the development of CAV.

There are several limitations to note. Our study is inherently biased as it is a retrospective review of the UNOS database. As such, we do not have information on selection criteria when a potential donor is available for any given recipient. Moreover, we are limited by the data and variables that are collected in the UNOS database. Chronic allograft vasculopathy is a multifactorial disease process, and some factors associated with CAV that were not assessed in our model include HLA matching and recipient use of calcium-channel blockers or cholesterol-lowering agents. Furthermore, the provided UNOS database does not allow us to resolve any missing data points, thereby resulting in a less impressive c statistic for our multivariate model. Also, we recognize the importance of a universal definition to CAV for the recipient population. However, both the UNOS and ISHLT organizations do not provide a standard method by which to detect CAV in recipients. Thus, the diagnosis of CAV is dependent on the individual transplant centers' screening algorithm and is self-reported.

In conclusion, we demonstrate that several donor factors are associated with development of CAV. We further identify an important relationship between donor and recipient age with respect to the development of CAV. Our models illustrate that advanced donor age confers a higher risk of CAV development regardless of recipient age and that older recipients are more sensitive to these effects. Recognition of this phenomenon suggests that recipients receiving advanced age donor hearts should be monitored more closely to minimize the potential for CAV development.

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Donor 10-19.9 years					
Recipient 18-29.9 years	913	613	399	185	9
Recipient ≥ 50 years	5775	4266	2875	1261	61
Donor ≥ 50 years					
Recipient 18-29.9 years	114	63	36	17	0
Recipient ≥ 50 years	2449	1638	883	264	3
Total	9251	6580	4193	1727	73
Numbers of Recipients at Risk					

Fig 1.

Kaplan-Meier age-matched analysis of recipient and donor populations for the development of chronic allograft vasculopathy (CAV) demonstrating that older donor age is associated with both the development of CAV and a negative effect on young recipients. Kaplan-Meier age-matched analysis depicting time to CAV development. As shown, young donors (10 to 19.9 years of age) conferred the lowest rate of CAV development among recipients when compared with old donors (≥ 50 years of age). Importantly, there is a significant ($p < 0.0001$) difference between the times to CAV development between a young donor and a young recipient compared with that of an old donor and a young recipient. Older donor age is associated with chronic rejection and development of CAV, and old donor age appears to have a greater negative effect in recipients.

Fig 2.

Percentage of recipients with chronic allograft vasculopathy (CAV) development after transplantation with a younger donor heart versus an older donor heart. Graphical representation of additional age-matched analysis demonstrating that at each permutation of donor and recipient age, recipients of older donor hearts had a higher percentage of chronic allograft vasculopathy development. These results suggest that an older donor heart may impart a baseline risk for the development of chronic allograft vasculopathy.

Table 1**Preoperative Demographics of Recipients**

Variable	N (%)
Recipient sex (n = 39,704)	
Male	30,633 (77.1)
Female	9,071 (22.9)
Recipient age, y (n = 39,704)	
Mean $\pm$ SD	51.0 $\pm$ 11.7
18–29.9	2,770 (7.0)
30–39.9	3,687 (9.3)
40–49.9	8,056 (20.3)
50–59.9	15,210 (38.3)
60–69.9	9,628 (24.2)
70–79.9	353 (0.9)
Recipient ethnicity (n = 39,667)	
White	31,658 (79.8)
Black	4,918 (12.4)
Other	3,091 (7.8)
Recipients with CAV (n = 39,704)	
No	27,990 (70.5)
Yes	11,714 (29.5)
Recipient history of cerebrovascular disease (n = 23,445)	
No	22,561 (96.2)
Yes	884 (3.8)
Recipient history of chronic obstructive pulmonary disease (n = 23,461)	
No	22,662 (96.6)
Yes	799 (3.4)
Recipient history of hypertension (n = 23,233)	
No	14,441 (62.2)
Yes	8,792 (37.8)
Recipient history of angina (n = 32,001)	
No	26,568 (83.0)
Yes	5,433 (17.0)
Recipient history of peripheral vascular disease (n = 23,356)	
No	22,525 (96.4)
Yes	831 (3.6)

CAV = chronic allograft vasculopathy; SD = standard deviation.

Table 2**Demographics of Donor Allografts to Adult Cardiac Transplant Recipients**

Variable	N (%)
Donor sex (n = 39,704)	
Male	27,910 (70.3)
Female	11,794 (29.7)
Donor age, y (n = 39,704)	
Mean $\pm$ SD	30.3 $\pm$ 12.3
0–9.9	118 (0.3)
10–19.9	9,563 (24.1)
20–29.9	11,614 (29.2)
30–39.9	8,231 (20.7)
40–49.9	6,966 (17.5)
50–59.9	2,904 (7.3)
60–69.9	297 (0.8)
70–79.9	11 (0.1)
Donor ethnicity (n = 39,610)	
White	29,882 (75.4)
Black	4,431 (11.2)
Other	5,297 (13.4)
Donor weight, kg (n = 36,226)	
Mean $\pm$ SD	77.1 $\pm$ 18.1
Donor death caused by trauma (n = 33,539)	
No	13,886 (41.4)
Yes	19,653 (58.6)
Donor cause of death (n = 39,598)	
Central nervous system tumor	332 (0.8)
Stroke	10,541 (26.6)
Head trauma	21,289 (53.8)
Other	7,436 (18.8)
Donor history of diabetes (n = 27,591)	
No	27,068 (98.1)
Yes	523 (1.9)
Donor active blood infection (n = 39,666)	
No	38,172 (96.2)
Yes	1,494 (3.8)
Donor with creatinine $\geq$ 1.5 (n = 39,704)	
No	32,699 (82.4)
Yes	7,005 (17.6)
Donor history of alcohol (n = 20,066)	
No	16,069 (80.1)
Yes	3,997 (19.9)

Variable	N (%)
Donor history of tobacco use (n = 27,433)	
No	18,623 (67.9)
Yes	8,810 (32.1)
Donor history of hypertension (n = 27,518)	
No	24,397 (88.7)
Yes	3,121 (11.3)
Donor history of IV drug abuse (n = 20,138)	
No	19,823 (98.4)
Yes	315 (1.6)
Donor CMV match (n = 15,373)	
No	7,045 (45.8)
Yes	8,328 (54.2)

CMV = cytomegalovirus; IV = intravenous; SD = standard deviation.

Table 3**Univariate Analysis of Donor and Recipient Variables Associated With Development of Chronic Allograft Vasculopathy**

Variable	CAV Negative (%)	CAV Positive (%)	p Value
Recipient sex (n = 39,704)			
Male (n = 30633)	21,257 (69.4)	9,376 (30.6)	<0.0001
Female (n = 9,071)	6,733 (74.2)	2,338 (25.8)	
Recipient age, y (n = 39,704)			
Mean ± SD	51.2 ± 12.0	50.5 ± 10.9	0.0001
18–29.9 (n = 2,770)	2,091 (75.5)	679 (24.5)	<0.0001
30–39.9 (n = 3,687)	2,612 (70.8)	1,075 (29.2)	
40–49.9 (n = 8,056)	5,325 (66.1)	2,731 (33.9)	
50–59.9 (n = 15,210)	10,494 (69.0)	4,716 (31.0)	
60–69.9 (n = 9,628)	7,178 (74.5)	2,450 (24.5)	
70–79.9 (n = 353)	290 (82.2)	63 (17.8)	
Recipient ethnicity (n = 39,667)			
White (n = 31,658)	22,013 (69.5)	9,645 (30.5)	<0.0001
Black (n = 4,918)	3,591 (73.0)	1,327 (27.0)	
Other (n = 3091)	2,361 (76.4)	730 (23.6)	
Donor sex (n = 39,704)			
Male (n = 27,910)	19,439 (69.7)	8,471 (30.3)	<0.0001
Female (n = 11,794)	8,551 (72.5)	3,243 (27.5)	
Donor age, y (n = 39,704)			
Mean ± SD	29.8 ± 12.1	31.6 ± 12.6	<0.0001
0–9.9 (n = 118)	89 (75.4)	29 (24.6)	<0.0001
10–19.9 (n = 9,563)	6,975 (72.9)	2,590 (27.1)	
20–29.9 (n = 11,614)	8,534 (73.5)	3,080 (26.5)	
30–39.9 (n = 8,231)	5,655 (68.7)	2,576 (31.3)	
40–49.9 (n = 6,966)	4,650 (66.8)	2,316 (33.2)	
50–59.9 (n = 2,904)	1,889 (65.1)	1,015 (34.9)	
60–69.9 (n = 297)	194 (65.3)	103 (34.7)	
70–79.9 (n = 11)	6 (54.6)	5 (45.4)	
Donor ethnicity (n = 39,610)			
White (n = 29,882)	20,535 (68.7)	9,347 (31.3)	<0.0001
Black (n = 4,431)	3,251 (73.4)	1,180 (26.6)	
Other (n = 5,297)	4,147 (78.3)	1,150 (21.7)	
Donor weight, kg (n = 36,226)			
Mean ± SD	77.0 ± 17.8	77.3 ± 18.6	0.2827
Donor death caused by trauma (n = 33,539)			
No (n = 13,886)	9,673 (69.7)	4,213 (30.3)	<0.0001
Yes (n = 19,653)	14,499 (73.8)	5,154 (26.2)	
Donor cause of death (n = 39,598)			

Variable	CAV Negative (%)	CAV Positive (%)	p Value
Central nervous system tumor (n = 332)	242 (72.9)	90 (27.1)	<0.0001
Stroke (n = 10,541)	7,099 (67.4)	3,422 (32.7)	
Head trauma (n = 21,289)	15,620 (73.4)	5,689 (26.6)	
Other (n = 7,436)	4,969 (66.8)	2,467 (33.2)	
Donor history of diabetes (n = 27,591)			
No (n = 27,068)	20,396 (73.4)	6,672 (24.6)	0.0694
Yes (n = 523)	376 (71.9)	147 (28.1)	
Donor active blood infection (n = 39,666)			
No (n = 38,172)	26,825 (70.3)	1,1347 (29.7)	<0.0001
Yes (n = 1,494)	1,148 (76.8)	346 (23.2)	
Donor with creatinine ≥ 1.5 (n = 39,704)			
No (n = 32,699)	22,723 (69.5)	9,976 (30.5)	<0.0001
Yes (n = 7,005)	5,267 (75.2)	1,738 (24.8)	
Donor history of alcohol (n = 20,066)			
No (n = 16,069)	11,153 (69.4)	4,916 (30.6)	<0.0001
Yes (n = 3,997)	2,645 (66.2)	1,352 (33.8)	
Donor history of tobacco use (n = 27,433)			
No (n = 18,623)	14,520 (78.0)	4,103 (22.0)	<0.0001
Yes (n = 8,810)	6,137 (69.7)	2,673 (30.3)	
Donor history of hypertension (n = 27,518)			
No (n = 24,397)	18,511 (75.9)	5,866 (24.1)	<0.0001
Yes (n = 3,121)	2,211 (70.8)	910 (29.2)	
Donor history of IV drug abuse (n = 20,138)			
No (n = 19,823)	13,625 (68.7)	6,198 (31.3)	0.3058
Yes (n = 315)	225 (71.4)	90 (28.6)	
Donor CMV match (n = 15,373)			
No (n = 7,045)	5,826 (82.7)	1,219 (17.3)	0.2768
Yes (n = 8,328)	6,942 (83.4)	1,386 (16.6)	

CAV = chronic allograft vasculopathy; CMV = cytomegalovirus; IV = intravenous; SD = standard deviation.

Table 4

Logistic Regression of Factors That May Influence the Development of Chronic Allograft Vasculopathy in Adult Cardiac Transplant Recipients

Factor	Odds Ratio	95% CI	p Value
Donor age (y)			<0.0001
0–19.9	1.0 ^a	...	
20–29.9	1.02	0.96–1.09	
30–39.9	1.27	1.18–1.36	
40–49.9	1.52	1.4–1.64	
≥50	1.72	1.56–1.9	
Donor ethnicity			<0.0001
White	1.0 ^a	...	
Black	0.92	0.86–0.99	
Other	0.76	0.7–0.81	
Donor sex			<0.0001
Male	1.0 ^a	...	
Female	0.75	0.71–0.79	
Donor diabetes			0.0007
No	1.0 ^a	...	
Yes	1.27	1.03–1.56	
Donor weight (kg)			0.003
<85 kg	1.0 ^a	...	
≥85 kg	1.09	1.03–1.16	
Donor tobacco use			<0.0001
No	1.0 ^a	...	
Yes	1.18	1.1–1.26	
Donor alcohol use			<0.0001
No	1.0 ^a	...	
Yes	0.99	0.91–1.07	
Donor hypertension			0.004
No	1.0 ^a	...	
Yes	1.1	1.01–1.21	
Donor IV drug use			<0.0001
No	1.0 ^a	...	
Yes	0.84	0.66–1.09	
Donor CMV match			<0.0001
No	1.0 ^a	...	
Yes	0.95	0.87–1.03	
Donor cause of death by trauma			0.5
No	1.0 ^a	...	
Yes	0.98	0.92–1.04	

Factor	Odds Ratio	95% CI	p Value
Donor creatinine			0.69
<1.5	1.0 ^a	...	
≥1.5	0.99	0.92–1.06	

^aReference group.

CI = confidence interval; CMV = cytomegalovirus; IV = intravenous.

Risk Ratios of Developing Chronic Allograft Vasculopathy of Donors 0 to 19.9 Years and ≥50 Years of Age When Matched to All Recipient Groups

Table 5

Recipient Age (y)	Recipients With CAV (%)		Risk Ratio of Developing CAV	95% CI	p Value
	Donor Age 0–19.9 Years	Donor Age ≥ 50 Years			
18–29.9	229 (25.1)	35 (30.7)	1.22	0.91–1.65	0.1955
30–39.9	281 (27.7)	54 (33.5)	1.21	0.95–1.54	0.1281
40–49.9	616 (31.1)	188 (38.5)	1.24	1.09–1.41	0.0018
≥50	1,493 (25.9)	848 (34.5)	1.34	1.25–1.43	<0.0001

CAV = chronic allograft vasculopathy; CI = confidence interval.