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Troponin I Levels from Donors Accepted for Pediatric Heart Transplantation do not Predict Recipient Graft Survival

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Abstract

Background—Troponin I (TnI) is often obtained during evaluation of a potential transplant donor heart. It is unclear whether elevations in donor TnI levels predict adverse outcomes and should thus preclude acceptance of a donor heart. The aim of this study was to examine whether TnI levels from donors accepted for pediatric heart transplantation predict graft failure.

Design/Methods—De-identified data on heart transplants performed in recipients ≤ 21 years old between 4/07–4/09 was provided by the Organ Procurement and Transplantation Network. Donor TnI level and recipient outcomes including survival without re-transplantation (graft survival) were examined for statistical correlation.

Results—Overall graft survival in 839 heart transplants was 81% at 2 years. 657 donors had at least one TnI level recorded prior to transplant, with values ranging from 0–50 ng/ml (median 0.1). There was no correlation between TnI level and graft status ($p=0.74$). An ROC curve showed no association between TnI and graft status ($AUC=0.51$; $p=0.98$), and there was no difference in graft survival ($p=0.60$) among quartiles of TnI (<0.04 , $0.04-<0.1$, $0.1-<0.35$, ≥ 0.35 ng/ml). 74 transplanted donors had a TnI ≥ 1 ; graft survival was not associated with TnI ≥ 1 (80%) vs TnI < 1 (80%) at 2 years ($p=0.93$). TnI values were also not associated with post-transplant hospital length of stay ($r=-0.06$; $p=0.10$).

Conclusions—In donor hearts accepted for pediatric heart transplantation, pre-procurement TnI elevations are not associated with increased graft failure. Many heart donors have elevated TnI levels whose significance remain unclear and should therefore be considered in the context of other clinical information.

Introduction

The assessment of a potential donor heart for transplantation in pediatrics is a critically important process. Although donor hearts are a limited resource, the pressure to accept a potential donor heart must be tempered by any identifiable donor risk factors for graft failure. The highest wait list mortality in heart transplantation is in infants and children less than 6 years of age (1), where the need for donor hearts far outstrips donor availability. A transplanted heart in a recipient of any age, however, must withstand numerous challenges, including the catecholamine surge surrounding brain death, the ischemic time between procurement and transplantation, and the hemodynamic demands of a sick transplant

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recipient. A biomarker which reflects donor heart quality could be quite useful in the timely evaluation of a potential donor heart.

Cardiac troponin I is a marker of myocardial damage which has been used for many years in the evaluation of acute coronary syndromes (2). The past several years have seen a dramatic increase in the number of centers requesting troponin I levels from a potential donor prior to acceptance of a donor heart for transplantation. Guidelines on how to interpret those values in this clinical scenario, however, are lacking. In this study, our objective is to determine whether troponin levels from a large, retrospective group of donors are associated with pediatric recipient graft survival.

Materials and Methods

Data Collection and Study Population

The United Network for Organ Sharing (UNOS) provided de-identified data on all heart transplants in recipients ≤ 21 years old ($n=847$) which took place between April 1, 2007 and April 30, 2009. Follow up is current through December 11, 2009. For any recipients who received two transplants within this time frame ($n=8$), we excluded the second transplant from analysis. The troponin I levels recorded in this data set represent the last available troponin I level reported to UNOS from a donor prior to transplantation. As troponin I levels were not a required data entry field for donor centers reporting to UNOS, all recorded donor troponin I values in this study were reported on a voluntary basis.

Statistical Methods

Descriptive results (including graphical displays) for all quantitative variables were reported as mean $\pm$ standard deviation (SD) for normally distributed data, or median with interquartile range (IQR) for data not normally distributed. Numbers (percentage) were reported for all qualitative variables. The two sample t-test was used to compare mean donor troponin I levels between graft status (alive or lost to follow up vs. dead or re-transplanted). A receiver operating characteristic (ROC) curve was also used to examine the relationship between donor troponin I levels and graft status. The primary outcome measure was graft survival duration (days), which was calculated from the date of transplantation to time of an event if the recipient status was listed as “dead” or “re-transplanted,” or censored if recipient status was listed as “lost to follow up” ($n=3$) or “alive” in the UNOS registry. Graft survival rates were estimated using Kaplan-Meier curves, and the association between graft survival and donor troponin I levels was assessed using the Log Rank test. Univariate Cox-Proportional Hazards models were used to estimate the hazard ratios (HR) and 95% confidence intervals that describe the relationships between graft failure and donor and recipient factors. Chi-square analysis was used to assess for association between hormonal resuscitation and donor troponin groups.

Secondary outcomes included length of stay from transplant to discharge (days), echocardiographic ejection fraction and shortening fraction after transplantation (%), and whether the recipient was treated for rejection within 1 year of transplantation (Yes/No). Pearson correlation was used to examine the relationships between donor troponin I levels and length of stay from transplant to discharge, as well as that between donor troponin I levels and post-transplant ejection fraction and shortening fraction. A two sample t-test was used to compare mean donor troponin I levels between subjects treated and not treated for rejection within 1 year of transplantation. A multiple Cox-regression model analysis was used to compare mean donor troponin I levels by various donor causes of death.

A two-sided p-value of <0.05 was considered statistically significant. All data were analyzed with a standard statistical software package, SAS (Statistical Analysis System, version 9.2).

Results

A total of 839 heart transplants in recipients ≤ 21 years old were analyzed; of those cases, 657 had a troponin I value recorded from the donor prior to organ procurement. Overall graft survival at 2 years was 81%. There was no difference in graft status between those with and those without a measured donor troponin I level ($p=0.75$). For those with a donor troponin I measurement, there was no correlation between donor troponin I level and graft status ($p=0.74$) (Fig1). 74 transplanted donors had a troponin I ≥ 1 ng/ml. Graft survival was virtually identical in those recipients with donor troponin I ≥ 1 ng/ml (79.8%) vs those with donor troponin I < 1 ng/ml (79.9%) at 2 years ($p=0.93$). There were also no differences in 2 year graft survival ($p=0.60$) within quartiles of donor troponin I levels (<0.04 , $0.04-0.1$, $0.1-0.35$, ≥ 0.35 ng/ml) (Fig2). An ROC curve showed no association between donor troponin I level and graft status ($AUC=0.51$; $p=0.98$) (Fig3).

Donor characteristics are listed in Table 1. Donor troponin I levels ($n=657$) ranged from 0-50 ng/ml, with a mean of 0.62 ± 2.78 and a median of 0.1 (IQR 0.04-0.35). As shown in Table 1, no subgroup of donor troponin I level (0.0-0.04, 0.04-0.1, 0.1-0.5, 0.5-1.0, 1.0-5.0, 5.0-50 ng/ml) had a hazard ratio significantly different from 1. In fact, the only donor characteristics that predicted a higher risk of graft failure were longer ischemic time, black ethnicity, absence of antihypertensive medication prior to cross-clamp, and absence of vasodilator medication prior to cross-clamp. Additionally, donor troponin I levels did not predict graft failure within any of the donor cause of death subgroups listed in the UNOS registry ($p=0.43$, 0.57, 0.82, and 0.55 for anoxia, cerebrovascular/stroke, head trauma, and other donor cause of death, respectively). There was no difference in whether hormonal resuscitation with thyroid hormone (triiodothyronine or thyroxine) was used for the donor between those with troponin levels ≥ 1 ng/ml and < 1 ng/ml ($p=0.77$).

Recipient characteristics and outcomes are listed in Table 2. The only recipient characteristics that were associated with risk of graft failure were ABO incompatibility, Class II panel reactive antibodies (PRA) $\geq 10\%$, and recipient need for mechanical support (i.e. ventricular assist device, intra-aortic balloon pump, and/or extracorporeal membrane oxygenation) (Table 2). There was no significant correlation between donor troponin I level and recipient length of stay from transplant to discharge ($r=-0.06$; $p=0.103$), ejection fraction after transplantation ($r=0.04$; $p=0.468$), or shortening fraction after transplantation ($r=0.02$; $p=0.791$). Donor troponin I level was also not associated with whether the recipient was treated (0.735 ± 1.97) vs. not treated (0.48 ± 0.86) for rejection within 1 year ($p=0.50$).

In recipients with the 20 highest donor troponin I values reported (range 3.37-50 ng/ml), 17 (85%) were alive without re-transplantation at the time of last follow-up. There was no statistical difference between the highest 20 troponin group and the entire cohort with respect to length of follow-up, recipient ejection fraction, shortening fraction, or length of stay from transplant to discharge ($p=0.88$, 0.95, 0.52, and 0.99). In the 74 recipients with a donor troponin I ≥ 1 ng/ml, 64 (86%) were alive without re-transplantation at the time of last follow-up. In this group of recipients with donor troponin I ≥ 1 ng/ml, we could not identify any characteristics that might have offset their higher troponins and thus protected them from worse outcomes as compared with those with a donor troponin I < 1 ng/ml (Table 3). In fact, pre-transplant donor ejection fraction was statistically lower in the high troponin group as compared with those whose donor troponin I was < 1 ng/ml (60.04 ± 10.5 vs. 63.46 ± 10.0 , $p=0.003$).

Discussion

In this retrospective analysis of all pediatric heart transplant recipients in the UNOS data registry for whom there are recorded donor cardiac troponin I levels from April 2007-April 2009, there was no correlation between donor troponin I levels and graft survival after transplantation. Indeed, grafts from donors with even the highest pre-procurement troponin I levels demonstrated survivability on par with the rest of the data set. In addition, we demonstrate that donor pre-procurement troponin I levels do not correlate with recipient length of stay from transplant to discharge, ejection fraction after transplantation, shortening fraction after transplantation, or whether the recipient was treated for rejection within 1 year of transplantation. The results of this large study examining the relationship between donor troponin levels and recipient outcomes in heart transplantation call into question the prognostic relevance of donor troponin levels.

Previous studies on this subject have been inconsistent. Several studies suggest that donor troponin levels may be used as a surrogate for functional assessment in potential donors. For example, Riou and colleagues prospectively linked increasing troponin T values with decreasing ejection fractions in 100 brain dead potential heart donors aged 16 to 68 years (3). In a prospective analysis of 79 potential heart donors, Venkateswaran and colleagues found that those with a troponin I greater than 1 ng/ml had higher central venous pressure and pulmonary arterial wedge pressure, lower cardiac index and fractional shortening, and worse wall motion score index than those with troponin I lower than 1 ng/ml (4). In aiming to establish troponin as a biochemical surrogate for donor cardiac function, the authors of this study noted that within their study group, nine of the 79 potential grafts were not retrieved due to poor function.

Boccheciampe and colleagues bolstered these findings, demonstrating that troponin I values were elevated in potential donors with ejection fractions below 50 percent and with segmental wall motion abnormalities compared with those with better functional parameters (5). However, this study also followed the recipients' outcomes and found no discernible differences in cardiac function one week after transplantation or, most importantly, in graft survival one year after transplantation between groups with varying troponin I values. Along a similar vein, Vijay and colleagues noted in a small cohort an increased incidence and severity of acute graft rejection in patients receiving hearts from donors with increased troponin T levels (6). However, a seven-year follow-up study demonstrated no differences in cardiac function or survival among the participants (7). Finally, when 139 heart donors in the California Transplant Donor Network were divided by pre-procurement troponin I levels (>1.0 ng/ml vs. ≤ 1.0 ng/ml), 30-day and one-year graft survival rates after transplantation were equal between the two groups (8).

However, conflicting evidence that links elevated troponins to poor outcomes exists as well. In Grant and colleagues' series of 19 infant heart donors and 18 infant heart recipients, five of eight grafts from donors with troponin I values greater than 3.2 ng/ml failed, and all grafts from donors with lower troponin I values survived (9). Troponin levels correlated with arrest time in the donor, and pathologic examination revealed myocardial necrosis suggestive of pre-procurement ischemic damage. In a larger retrospective study of 118 brain dead donors above ten years of age, Potapov and colleagues found that elevations in both donor troponin I and troponin T were associated with early graft failure after transplantation (10).

Although somewhat contradictory, these prior studies and ours together demonstrate that elevated donor troponin values present a common and unresolved conundrum in heart procurement. Cardiac troponin I is a highly sensitive and specific biomarker for myocardial damage. It has become essential in the diagnosis of acute myocardial infarction (11), and its

presence is often observed in cases of myocardial stunning, myocardial reperfusion, and acute myocarditis (12). Serum cardiac troponins also rise with cardiopulmonary resuscitation (13,14), cardioversion (15), and significant blunt trauma (16). A root cause of elevated troponin levels in potential heart donors that is less well understood, but likely very significant in our pediatric population, is brain death itself. Researchers and clinicians have long observed the occurrence of decreasing cardiac function to varying degrees following brain death in patients (3,17) that may often preclude heart procurement. It is thought that impending brain death leads to a catecholamine surge from the stressed sympathetic neuroendocrine system that impairs function in a number of ways. This surge has been observed in human (18,19) and animal (20-22) studies, and has been linked to poor hemodynamic stability and cardiac function (20), decreased coronary blood flow (23), cardiomyocyte damage (24), as well as increased troponin production and turnover (25). Whether troponin elevations and impaired function reflect irreversible cardiac damage in potential donors who suffer a non-cardiac arrest remains a point of much debate (3,26-28).

One must be cautious not to conclude from these retrospective results that an elevated troponin I level is irrelevant in the evaluation of a potential donor heart. While donor troponin I levels did not predict graft survival in this cohort of subjects that went on to transplantation, we do not know what would have happened to the other potential donors with elevated donor troponin I levels that did not go on to transplantation. It is our practice to request further details when an otherwise acceptable donor has an elevated troponin level. Has the reported value been verified? What is the trend in troponin levels? What was the timing of troponin verification with respect to the determination of brain death? Perhaps, as suggested in a recent review of the role of biomarkers in assessment of the potential heart donor, we will soon have additional biomarkers that can be interpreted in combination with troponin levels to better discriminate between acceptable and unacceptable donor hearts (29).

Limitations

The main limitations of this study relate to its retrospective design. This cohort only includes outcome data and troponin values from donor hearts that went on to transplantation. While clinical information including troponin values from donor hearts that are declined is available, by definition there is no transplant outcome information in that situation. Selection bias may certainly be one reason that the outcomes from our donors with the highest recorded troponin values is no different from the rest of the cohort.

Furthermore, the troponin I values provided by UNOS were all performed by the laboratory affiliated with the care of the donor, rather than a central laboratory. The normal and detectable ranges of troponin I may vary by the laboratory in which it is performed as well as the specific assay that is used. In this study, we have compared troponin I levels between graft survival and failure groups as if they were all performed by identical methodologies. We did not have access to the specific methodologies utilized by each laboratory. Whenever possible, however, it is important to assess the normal range and precision in a particular laboratory of the troponin assay used, rather than solely relying on the absolute value of troponin level reported.

Conclusions

In donor hearts accepted for pediatric heart transplantation, pre-procurement donor troponin I elevations are not associated with increased graft failure. As demonstrated by this large, national cohort, many heart donors have elevated troponin I levels whose significance remains unclear. Additional studies looking at the timing of and serial trends in donor troponin ascertainment may provide further guidance to the clinician who evaluates potential

donor heart quality. Taken in isolation, an elevated troponin I level in a potential heart donor may not be enough to preclude consideration for heart transplantation. Rather, such data should be considered in the context of other clinical information.

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Figure 1. Mean Troponin I Levels by Graft Status

Figure 2. Graft Survival Stratified by Donor Troponin I Quartiles

Figure 3. Receiver Operator Characteristic Curve for Troponin I Level and Graft Status

Table 1
Donor Characteristics and Hazard Ratios for Graft Loss

Factors	Results	HR (95% CI)	P value
Age (Years), N=839	11±11.25	1.00(0.98-1.01)	0.6547
Gender			
Female	341(41%)	reference	
Male	498(59%)	1.04(0.71-1.53)	0.8216
Ethnicity			
Black	181(22%)	1.58(1.01-2.47)	0.0443
Hispanic	189(23%)	1.33(0.84-2.10)	0.2296
White	448(53%)	reference	
Other	21(3%)	0.43(0.06-3.08)	0.3983
Decreased Donor Cause of Death			
Anoxia	217(26%)	0.62(0.25-1.53)	0.3002
Cerebrovascular/Stroke	69(8%)	0.94(0.34-2.59)	0.9041
CNS Tumor	7(1%)	0.91(0.11-7.53)	0.9271
Head Trauma	506(60%)	0.86(0.37-1.98)	0.7225
Other	40(5%)	reference	
Troponin I Group			
0.0 Thru <0.04	160(24%)	reference	
0.04 Thru <0.1	164(25%)	0.98(0.53-1.79)	0.9368
0.1 Thru <0.5	203(31%)	0.99(0.55-1.76)	0.9662
0.5 Thru < 1.0	56(9%)	1.06(0.46-2.40)	0.8970
1.0 Thru < 5.0	62(9%)	0.91(0.40-2.07)	0.8272
5.0 Thru 50	12(2%)	1.24(0.29-5.31)	0.7708
History of Hypertension			
Yes	19(2%)	1.27(0.40-4.00)	0.6818
No	818(98%)	reference	
Antihypertensives Pre-cross Clamp			
Yes	128(15%)	reference	
No	709(85%)	2.55(1.24-5.24)	0.0109
Vasodilators Pre-cross Clamp			
Yes	105(13%)	reference	
No	731(87%)	3.31(1.35-8.13)	0.0089
Pre-recovery Synthetic Antidiuretic Hormone (DDAVP)			
Yes	148(18%)	1.27(0.81-1.99)	0.3044
No	689(82%)	reference	
Pre-recovery Arginine (Vasopressin)			
Yes	521(62%)	0.73(0.50-1.06)	0.0998
No	315(38%)	reference	
Pre-recovery Inotropic agents			
Yes	412(49%)	1.07(0.74-1.56)	0.7103

Factors	Results	HR (95% CI)	P value
No	427(51%)	reference	
Troponin I (ng/ml), N=657	0.1(0.04-0.35)	1.01(0.95-1.08)	0.7420
Pre-Transplant Donor EF (%), N=806	63±10.70	1.00(0.98-1.02)	0.7278
Ischemic Time (Hours), N=809	4±1.24	1.16(1.01-1.33)	0.0357

Results are expressed as mean ± standard deviation or median (inter quartile range) for continuous variables; n (%) for categorical variables.
Definition of abbreviations: HR=hazard ratio; CI=confidence interval; PRA=panel reactive antibodies; EF=ejection fraction

Table 2
Recipient Characteristics and Hazard Ratios for Graft Loss

Factors	Results	HR (95%CI)	P value
Age (Years), N=839	8±7.35	0.99(0.96-1.01)	0.3902
Gender			
Female	370(44%)	reference	
Male	469(56%)	0.84(0.58-1.22)	0.3631
Ethnicity			
Black	181(22%)	1.12(0.71-1.78)	0.6276
Hispanic	147(18%)	0.85(0.49-1.48)	0.5620
Other	63(8%)	1.51(0.79-2.87)	0.2141
White	448(53%)	reference	
Status at Transplant			
1A	653(78%)	1.34(0.62-2.91)	0.4526
1B	124(15%)	1.38(0.57-3.35)	0.4719
2	62(7%)	reference	
PRA Percent-Class I			
≥ 10%	113(15%)	1.28(0.76-2.16)	0.3527
< 10%	632(85%)	reference	
PRA Percent-Class II			
≥ 10%	87(12%)	1.71(1.01-2.89)	0.0450
< 10%	627(88%)	reference	
ABO Compatible			
Yes	811(97%)	reference	
No	28(3%)	2.65(1.29-5.46)	0.0081
Need for mechanical support			
Yes	176(21%)	1.82(1.21-2.73)	0.0041
No	663(79%)	reference	
Waiting Time From Initial Listing To Transplant (Days), N=839	45 (15 – 101)	1.00(1.00-1.00)	0.6499
Patient status			
Dead	100(12%)		
Living	724(86%)		
Lost To Follow Up	3(0%)		
Re-Transplanted	12(1%)		
Treated for rejection within 1 year			
Yes	44(34%)		
No	87(66%)		
Patient Survival Time (Days), N=839	357 (186 – 422)		
Length Of Stay After Transplant (Days), N=815	17 (11 – 29)		
Post-Transplant EF (%), N=368	65 ± 11.23		
Post-Transplant SF (%), N=360	38 ± 6.82		

Results are expressed as mean ± standard deviation or median (inter quartile range) for continuous variables; n (%) for categorical variables. Need for mechanical support prior to transplant is defined as “yes” if the recipient was on LVAD, LVAD+RVAD, RVAD, ECMO, and/or IABP prior to

transplant. Definition of abbreviations: HR=hazard ratio; CI=confidence interval; PRA=panel reactive antibodies; EF=ejection; SF=shortening fraction; LVAD=left ventricular assist device; RVAD=right ventricular assist device; ECMO=extracorporeal membrane oxygenation; IABP=intra-aortic balloon pump

Table 3
Comparison of Low and High Donor Troponin Groups

Variables	Category	Troponin Level		P value
		< 1 ng/ml	≥ 1 ng/ml	
History of Hypertension	N	571(98%)	70(95%)	0.0624
	Y	10(2%)	4(5%)	
Antihypertensives Pre-cross Clamp	N	498(85%)	60(81%)	0.3257
	Y	85(15%)	14(19%)	
Vasodilators Pre-cross Clamp	N	508(87%)	63(85%)	0.6039
	Y	74(13%)	11(15%)	
Pre-recovery Synthetic Antidiuretic Hormone(DDAVP)	N	477(82%)	61(82%)	0.8972
	Y	106(18%)	13(18%)	
Pre-recovery Arginine (Vasopressin)	N	220(38%)	31(42%)	0.4331
	Y	363(62%)	42(58%)	
Pre-recovery Inotropic agents	N	303(52%)	35(47%)	0.4484
	Y	280(48%)	39(53%)	
Ischemic Time (Hours)		3.58±1.25	3.59±1.24	0.7104
Pre-Transplant Donor EF (%)		63.46±10.01	60.04±10.48	0.0030

Results are expressed as mean ± standard deviation for continuous variables; n (%) for categorical variables. Definition of abbreviations: EF=ejection fraction