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ASSOCIATION BETWEEN CIRCULATING ANTI-HLA IgG ANTIBODIES AND ADVERSE EVENTS IN HEART TRANSPLANT RECIPIENTS

A.Yu. Goncharova[1], Yu.A. Kachanova[1], N.N. Koloskova[1], O.E. Gichkun[1, 2], A.O. Shevchenko[1–3]

[1] Shumakov National Medical Research Center of Transplantology and Artificial Organs, Moscow, Russian Federation

[2] Sechenov University, Moscow, Russian Federation

[3] Pirogov Russian National Research Medical University, Moscow, Russian Federation

Objective: to study the relationship between circulating anti-HLA antibodies and the incidence of adverse events (death and retransplantation) and to evaluate the effectiveness of targeted therapies for graft dysfunction. **Materials and methods.** A retrospective study was conducted among heart transplant recipients hospitalized with signs of circulatory failure (graft dysfunction). All patients underwent coronary angiography, endomyocardial biopsy, and serological testing for anti-HLA IgG antibodies at baseline and, when applicable, after treatment. All heart transplant recipients underwent endomyocardial biopsy with examination of six tissue samples using histological and immunohistochemical techniques, and coronary angiography. Anti-HLA IgG levels in serum were measured using a Luminex device. Follow-up anti-HLA IgG testing was performed only in patients with initially detectable antibodies and after administration of specific therapies for presumed graft rejection. **Results.** The study included 362 heart transplant recipients observed at Shumakov National Medical Research Center of Transplantology and Artificial Organs between January 2018 and November 2024. Participants were aged 18–72 years (mean 48.1 ± 1.3 years) with a mean post-transplant follow-up of 1343.6 ± 125.1 days (95% CI 1218.5–1408.6), comprising 69 females and 293 males. Anti-HLA IgG antibodies were detected in 111 recipients (30.7%). Univariate analysis identified significant associations between adverse events and: repeat heart transplantation ($p = 0.005$), perioperative use of mechanical circulatory support ($p < 0.003$), age under 46 years at hospitalization ($p = 0.023$), and anti-HLA II maxMFI above 5000 ($p = 0.042$). Regression analysis adjusted for anti-HLA II levels showed that only initially elevated anti-HLA II maxMFI levels (>5000) and the persistence of any anti-HLA II levels after etiotropic treatment were associated with the risk of adverse events. **Conclusion.** In heart recipients hospitalized with signs of circulatory failure due to graft dysfunction, the presence of anti-HLA II maxMFI titers above 5000 at baseline, as well as residual anti-HLA II titers after etiotropic treatment for antibody-mediated rejection, are independent predictors of adverse events, including retransplantation and death.

Keywords: heart transplantation, anti-HLA, antibody-mediated rejection.

INTRODUCTION

Heart transplantation (HT) remains the gold standard treatment for end-stage chronic heart failure (CHF) when optimal medical therapy is ineffective and/or radical surgical intervention on the native heart is not feasible. Despite significant advances in diagnostic techniques and therapeutic strategies for heart transplant recipients, graft dysfunction remains a major challenge in modern transplantology. It is a leading cause of recurrent hospitalizations and is associated with an increased risk of adverse clinical outcomes [1].

The human leukocyte antigen (HLA) complex was first identified by French immunologist Jean Dausset in the 1950s as a group of proteins expressed on white blood cells. It was later discovered that antibodies to HLA (anti-HLA antibodies) play a central role in graft rejection in kidney transplant recipients [2]. In the 1960s, Paul Terasaki and David McClelland developed the complement-dependent lymphocyte cytotoxicity (CDC) assay. This technique enabled the detection of anti-HLA antibodies [3]. Subsequently, various other techniques for detecting anti-HLA have been developed [4].

The HLA system comprises three classes of molecules: class I (loci A, B, and C), class II (loci DR, DQ, and DP), and class III, which includes genes not directly involved in antigen presentation. All are encoded within different regions of chromosome 6. HLA expression on the cell surface enables immune recognition of "self" and "non-self" cells. Class I HLA molecules are expressed on all nucleated cells, whereas the expression of class II HLA molecules is restricted to B cells, activated T cells,

Corresponding author: Olga Gichkun. Address: 1, Shchukinskaya str., Moscow, 123182, Russian Federation. Phone: (499) 190-38-77. E-mail: Gichkunoe@yandex.ru

and antigen-presenting cells. Expression of HLA class I and II on allograft vascular endothelium may explain the rejection that occurs in the presence of donor-specific antibodies (DSAs) [5].

Antibody-mediated allograft injury occurs primarily through activation of the complement system. Binding of antibodies to HLA antigens triggers C1q activation and initiates the complement cascade, ultimately leading to tissue damage. In addition, complement-independent mechanisms also contribute to graft injury. DSAs can recruit inflammatory cells via Fc receptor interactions, promoting the release of pro-inflammatory mediators. Antibodies directed against major histocompatibility complex antigens can therefore induce both direct endothelial injury and indirect damage mediated by complement fixation or attraction of inflammatory cells to Fc receptors [6]. As a result, cellular inflammation, thrombosis, hemorrhage, and lysis cause cardiac allograft dysfunction [7].

Despite advances in therapeutic approaches, heart transplant dysfunction and its relationship with circulating anti-HLA antibodies remain an area of active investigation. The aim of this study was to assess the association between serum anti-HLA antibodies and the incidence of adverse outcomes – namely death and retransplantation – and to evaluate the effectiveness of targeted treatment strategies for transplant-related cardiac dysfunction.

MATERIALS AND METHODS

The results obtained in this study were based on the examination of heart recipients who underwent orthotopic heart transplantation (OHT) at Shumakov National Medical Research Center of Transplantology and Artificial Organs. All patients underwent routine endomyocardial biopsy with analysis of six myocardial samples using histological and immunohistochemical methods, coronary angiography, and assessment of serum anti-HLA IgG antibodies using Luminex technology. Repeat testing for anti-HLA IgG antibodies was performed only in patients with initially detected antibodies after completion of targeted anti-rejection therapy.

In the perioperative and early postoperative periods, all recipients received induction therapy with basiliximab followed by triple-drug maintenance immunosuppression consisting of tacrolimus, mycophenolate mofetil/mycophenolic acid, and methylprednisolone. Six months after HT, patients were transitioned to dual-drug maintenance immunosuppression with tacrolimus and mycophenolate mofetil or mycophenolic acid.

Upon the development of heart transplant dysfunction, patients were routinely admitted for inpatient care at Shumakov National Medical Research Center of Transplantology and Artificial Organs. All heart transplant recipients underwent a comprehensive diagnostic evaluation, including endomyocardial biopsy (with analysis of six samples using histological and immunohistochemical methods), coronary angiography, and assessment of serum anti-HLA IgG levels. Antibody levels were quantified as mean fluorescence intensity (maxMFI) using multiplex analysis on the Luminex® platform.

Repeat measurements of anti-HLA IgG were performed only in patients in whom antibodies were initially detected, following targeted treatment for transplant rejection. Treatment strategies were implemented in accordance with the 2023 clinical guidelines and included pulse corticosteroid (methylprednisolone) therapy, intravenous immunoglobulin, therapeutic plasma exchange, and rituximab infusions [8].

In cases of cardiac allograft vasculopathy (CAV), percutaneous coronary intervention was performed, with or without stent implantation in the affected coronary arteries.

The risk of adverse events was assessed using multiple study factors, such as age, sex, pre-transplant diagnosis, the need for mechanical circulatory support (MCS), and a history of preoperative heart transplant rejection in re-transplant patients. MCS modalities included biventricular assist devices combined with a membrane oxygenator (ECMO) and long-term MCS systems, such as AVK-N and HeartMate 3.

After data collection was completed, all patient data were consolidated into a single electronic database. Statistical analysis was performed using SPSS version 26 (IBM SPSS Inc., USA). Parametric variables are presented as the mean ± standard deviation (M ± SD), while nonparametric variables are reported as the median (Med) and interquartile range (IQR; Q1–Q3). Comparisons between dependent samples were performed using the Wilcoxon signed-rank test, and comparisons between independent samples were conducted using the Mann–Whitney U test.

The prognostic value of anti-HLA antibody levels for the development of adverse events was evaluated using univariate logistic regression analysis. Accuracy of prediction was assessed using the Hosmer–Lemeshow test, and model performance was quantified using the Nagelkerke coefficient of determination. The statistical significance of the model was assessed using Wald's chi-square test. For all criteria, the critical level of significance was set at 5%, i.e., the null hypothesis was rejected at $p < 0.05$.

RESULTS

This study included blood samples from 362 HT recipients who underwent OHT at Shumakov National Medical Research Center of Transplantology and Artificial Organs between 2010 and 2024. The mean follow-up duration was 1343.6 ± 125.1 days (95% CI: 1218.5–1408.6). The mean age of the recipients was 48.1 ± 1.3 years (range: 18–72 years). Of the total cohort, 69 patients were female (19.1%) and 293 were male (80.9%).

Indications for HT included end-stage CHF due to ischemic cardiomyopathy (ICM) in 116 patients (32.0%), dilated cardiomyopathy (DCM) in 211 patients (58.3%), restrictive cardiomyopathy (RCM) in 5 patients (1.4%), and hypertrophic cardiomyopathy (HCM) in 10 patients (2.8%). In addition, heart retransplantation due to post-transplant graft dysfunction with manifestations of circulatory failure was performed in 15 cases (4.1%). Other indications included peripartum cardiomyopathy (3, 0.8%) and arrhythmogenic right ventricular cardiomyopathy (2, 0.6%). MCS as a bridge to HT was required in 80 patients (22.1%).

Episodes of HT rejection, both cellular and antibody-mediated forms, were evaluated prior to laboratory assessment of anti-HLA antibody levels. A history of rejection episodes was identified in 65 recipients (17.9%), whereas 297 patients (82.1%) had no documented rejection.

Coronary artery vasculopathy of the transplanted heart (CAVTH), as determined by coronary angiography, was identified in 84 heart recipients (23.2%). Histological examination of endomyocardial biopsies revealed acute cellular rejection in 48 patients, with 42 (11.6%) exhibiting R1G-grade, 4 (1.1%) R2G-grade, and 2 (0.6%) R3G-grade rejection. Immunohistochemical examination identified acute antibody-mediated rejection (AMR) in 67 recipients: AMR-1 (I+) in 44 patients (12.2%), AMR-1 (H+) in 15 patients (4.1%), and AMR-2 in 8 patients (2.2%).

The general characteristics of the study population are summarized in Table 1.

Table 1

Characteristics of the study population (n = 362)

Characteristic		Value
Sex:	Male, n (%) Female, n (%)	293 (80.9%) 69 (19.1%)
Age (years) mean ± SD CI		 48.1 ± 1.3 (CI 95% 46.7–49.4)
Initial diagnosis, n (%): ICM DCM RCM HCM CTD Peripartum CM Arrhythmogenic right ventricular CM		 116 (32%) 211 (58.3%) 5(1.4%) 10 (2.8%) 15(4.1%) 3(0.8%) 2(0.6%)
Use of MCS, n (%)		80 (22.1%)
Episodes of transplant rejection prior to study, n (%)		65 (17.9%)
CAV, n (%)		84 (23.2%)
Acute cellular rejection, n (%): R0G R1G R2G R3G		 314 (86.7%) 42 (11.6%) 4 (1.1%) 2 (0.6%)
Acute antibody-mediated rejection, n (%): pAMR-0 pAMR-1 (I+) pAMR-1 (H+) pAMR-2		 295 (81.5%) 44 (12.2%) 15 (4.1%) 8 (2.2%)
Presence of anti-HLA, n (%)		111 (30.7%)

Abbreviations: SD, standard deviation; CI, confidence interval; ICM, ischemic cardiomyopathy; DCM, dilated cardiomyopathy; RCM, restrictive cardiomyopathy; HCM, hypertrophic cardiomyopathy; CTD, connective tissue dysplasia; CM, cardiomyopathy; MCS, mechanical circulatory support; CAV, cardiac allograft vasculopathy.

The study found that patient sex and a history of rejection episodes prior to OHT in patients with HT dysfunction requiring heart retransplantation were not associated with a worse post-transplant prognosis ($p > 0.05$). In contrast, the use of MCS prior to OHT was associated with a significantly increased risk of adverse postoperative events ($p < 0.003$).

The analysis revealed that recipients with end-stage heart failure (HF) secondary to heart transplant dysfunction had a significantly higher risk of adverse events compared with patients with other preoperative heart pathologies ($p = 0.005$).

Survival analysis revealed that recipients aged 46–72 years had a significantly higher survival rate than younger recipients aged 18–45 years ($p = 0.023$).

Elevated serum levels of anti-HLA IgG antibodies were found to have a statistically significant negative impact on post-transplant prognosis ($p = 0.004$). This part of the study considered anti-HLA antibodies of all classes (both class I and class II) without separately assessing the prevalence of antibodies against specific loci (A, B, C for Class I; DR, DQ, DP for Class II).

A summary of the associations between adverse event risk and the evaluated clinical factors is presented in Fig. 1.

According to the study results, transplant CAV did not demonstrate a statistically significant link to the risk of adverse events ($p > 0.05$). In contrast, ACR (specifically R1G-grade rejection) was associated with a poorer prognosis ($p = 0.012$).

Evaluation of AMR did not reveal a statistically significant impact on prognosis for HT recipients at the time of analysis; however, a trend toward significance was observed ($p = 0.073$).

The prognostic effects of different types of ACR and AMR in HT recipients are illustrated in Fig. 2.

After testing serum anti-HLA antibody levels, all HT recipients were stratified into two subgroups: the first consisted of patients with detectable anti-HLA IgG antibodies (n = 111, 30.7%), while the second subgroup included recipients without detectable DSAs (n = 251, 69.3%).

A comparative analysis between the two subgroups is presented in Table 2.

The two study groups did not differ significantly with respect to demographic characteristics (age and sex) or the incidence of post-transplant complications (acute HT rejection and CAV ($p > 0.05$). However, the use of MCS methods prior to transplantation was significantly more frequent among recipients with detectable serum anti-HLA antibodies, whereas a higher proportion of patients without anti-HLA antibodies had end-stage HF due to dilated cardiomyopathy in the pre-transplant period ($p = 0.02$).

The presence of anti-HLA class I antibodies was not associated with the risk of post-transplant complications, likely reflecting their lower prevalence in the study cohort. In addition, circulating anti-HLA class II antibodies did not show a statistically significant association with the development of ACR or CAV ($p > 0.05$). In contrast, elevated anti-HLA class II antibody levels were significantly associated with the occurrence of AMR after HT, with AMR patients exhibiting higher maxMFI values compared to non-AMR patients ($p = 0.004$) (Fig. 3).

To assess treatment effectiveness, serum anti-HLA class II antibody levels were measured before and after targeted therapy for HT rejection. Changes in anti-HLA class II levels before and after treatment are presented in Fig. 4.

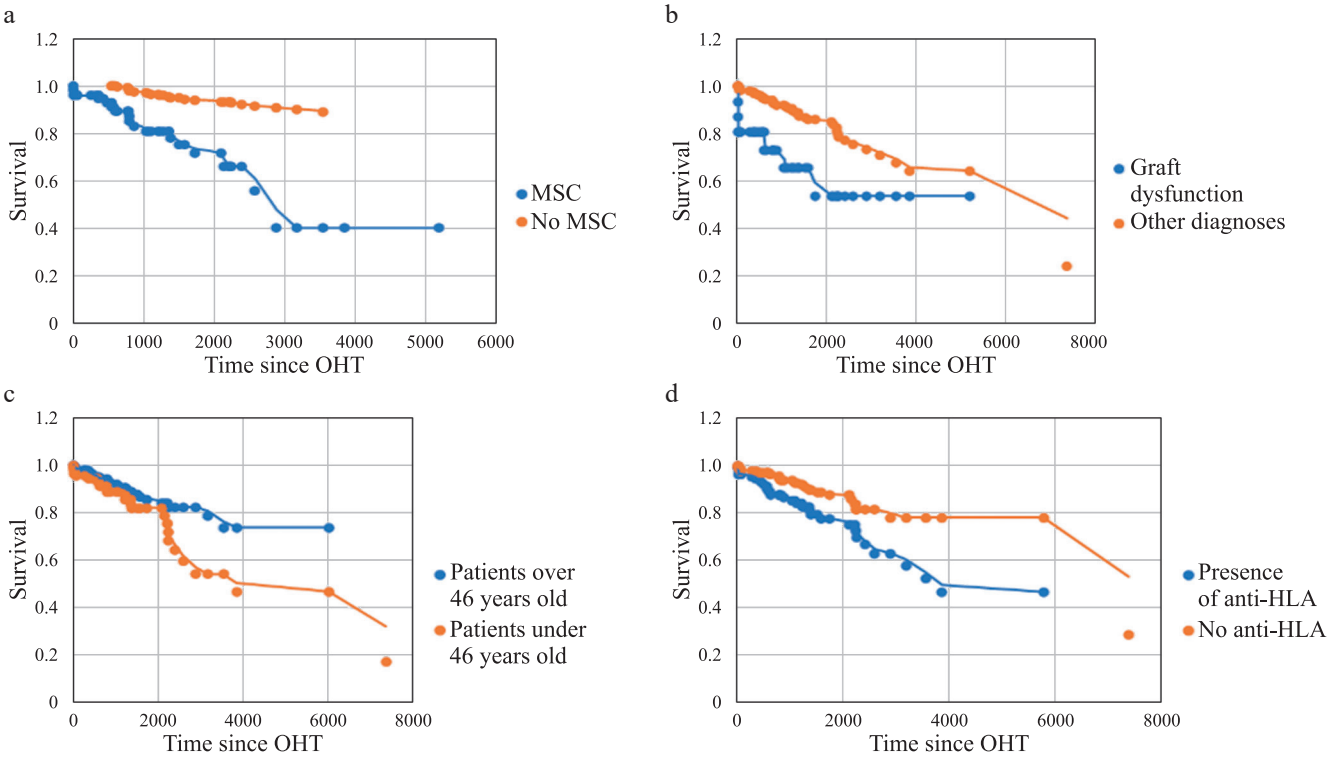

Fig. 1. Comparative analysis of event-free survival depending on MSC (a), graft dysfunction (b), age (c) and presence of anti-HLA antibodies of both classes (d). Note: MCS, perioperative mechanical circulatory support; OHT, orthotopic heart transplantation

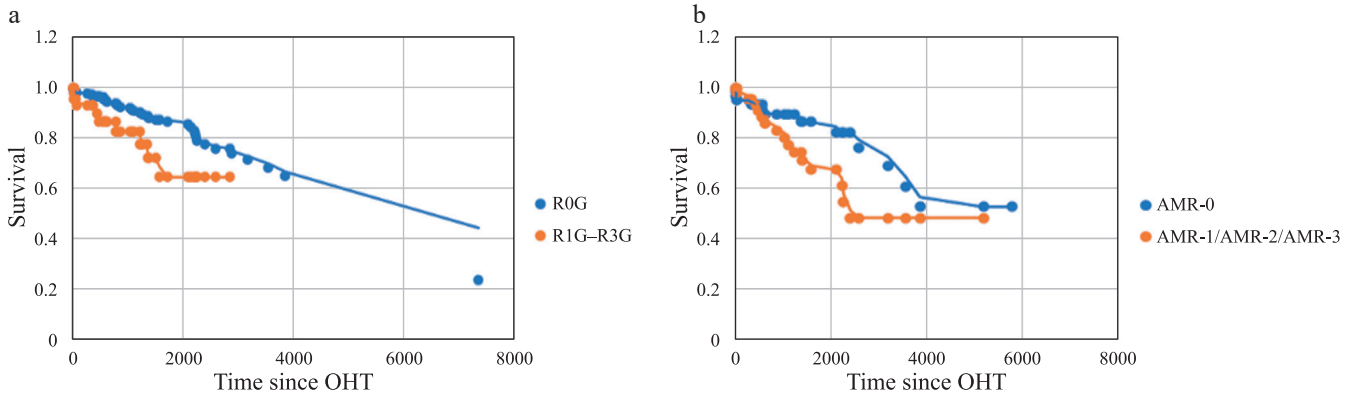

Fig. 2. Comparative analysis of survival without adverse events depending on acute cellular rejection (a) and acute antibody-mediated rejection of heart transplants (b)

Recipients with elevated pre-treatment anti-HLA class II maxMFI levels (>5000) exhibited a significantly poorer prognosis compared with patients whose anti-HLA class II maxMFI <5000 (p = 0.042), as illustrated in Fig. 5.

During treatment, patients were categorized into three cohorts: those in whom anti-HLA class II IgG levels became undetectable after treatment (45 patients, 40.5%), those in whom levels decreased but remained detectable (41 patients, 37%), and those in whom the anti-HLA class II IgG levels did not change during treatment (25 patients, 22.5%).

After treatment for HT rejection, serum anti-HLA class II levels were reassessed and correlated with clini-

Table 2

Comparative characteristics of heart recipients with and without anti-HLA antibodies in their blood serum

Characteristic	Anti-HLA positive	Anti-HLA negative	p-value
Number of patients, n	111	251	
Sex (Male), n (%)	78.4%	82.1%	0.25
Age (years) mean ± SD CI	 45.7 ± 1.3 (CI 95% 47.6–50.6)	 49.1 ± 0.78 (CI 95% 43.1–48.3)	 0.12
Cellular rejection, n (%)	27%	8.8%	0.15
Antibody-mediated rejection, n (%)	47.7%	7.7%	0.3
CAV, n (%)	18%	23.1%	0.45
Diagnosis prior to OHT (DCM), n (%)	53.2%	60.3%	0.02
MCS prior to OHT, n (%)	28.8%	19.1%	0.02

Note: SD, standard deviation; CI, confidence interval; CAV, cardiac allograft vasculopathy; OHT, orthotopic heart transplantation; DCM, dilated cardiomyopathy; MCS, mechanical circulatory support.

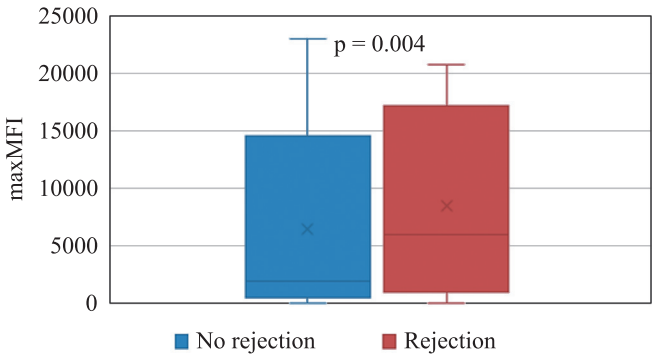

Fig. 3. Comparative analysis of maxMFI of anti-HLA class II in heart recipients with and without antibody-mediated rejection

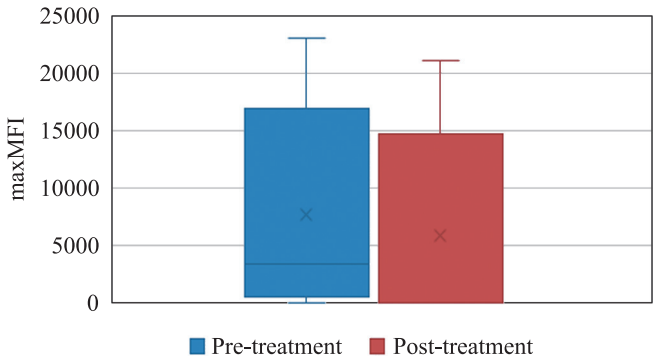

Fig. 4. Dynamics of anti-HLA class II maxMFI levels before and after targeted treatment for acute rejection

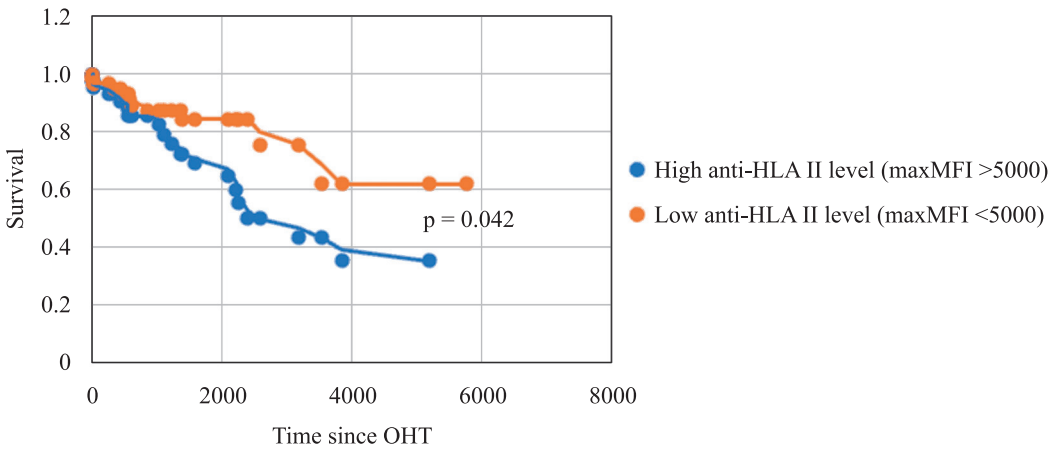

Fig. 5. Risk of adverse events in heart transplant recipients with high and low pre-treatment serum anti-HLA class II IgG levels

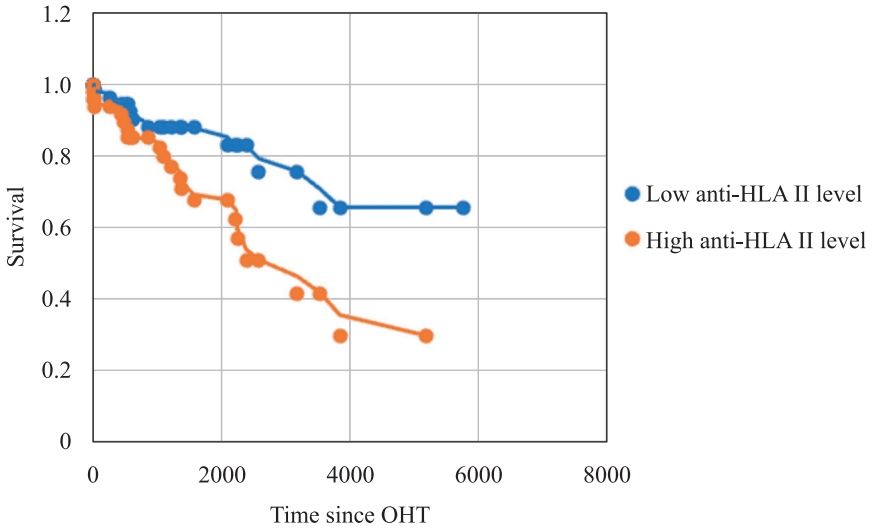

Fig. 6. Comparative analysis of event-free survival depending on post-treatment anti-HLA class II IgG levels

cal outcomes. A favorable dynamic in anti-HLA class II levels was observed, accompanied by a significant reduction in the risk of adverse events ($p = 0.017$), as shown in Fig. 6.

DISCUSSION

The results of this study confirm the significant role of anti-HLA class II antibody levels in the development of AMR and their negative impact on adverse events in heart recipients. Elevated anti-HLA class II levels were strongly associated with AMR ($p = 0.004$), corroborating the findings of Shemakin et al. [9]. Recipients with high pre-treatment anti-HLA class II values (maxMFI >5000) exhibited a poorer prognosis compared with those with lower levels ($p = 0.042$).

Measurement of anti-HLA antibodies provides a valuable tool for evaluating the effectiveness of therapy for AMR, complementing morphological assessments. In our cohort, treatment for HT rejection resulted in a favorable reduction of anti-HLA class II levels in serum, which was accompanied by a significant decrease in the risk of adverse events ($p = 0.017$).

The study further identified cohorts of patients with complete or partial elimination of anti-HLA class II antibodies following treatment, as well as a cohort in which antibody levels remained unchanged. These findings once again underscore the importance of thorough pre-treatment evaluation of HT recipients and the need for individualized selection of therapy for AMR. Our results are consistent with those of Clerkin et al., who reported that patients with anti-HLA were 151% more likely to experience graft loss – due to death or retransplantation – compared with patients without detectable anti-HLA [9]. Similarly, Smith et al. reported no direct correlation between anti-HLA and CAV in their cohort; however, when examining causes of death, a substantial proportion of patients with anti-HLA died as a result of CAV or transplant rejection [10].

In our study, patients received targeted therapy for transplant rejection and high immunological risk, consistent with the approaches described by Marco et al. [11], who proposed a diagnostic and treatment algorithm for patients with anti-HLA antibodies, and by Kamath et al. [12]. In addition, we assessed the impact of several factors on long-term outcomes in HT recipients, including the use of MCS, the underlying pre-transplant cardiac pathology leading to end-stage heart failure, and patient age.

Our results showed that prior use of MCS significantly increased the risk of post-transplant adverse events. Moreover, recipients with a pre-transplant diagnosis of "heart transplant dysfunction" as the cause of end-stage HF were more likely to experience postoperative complications. Younger recipients, and those with circulating anti-HLA IgG antibodies have a poorer prognosis compared with patients without such baseline data. The observed effect of MCS on post-transplant outcomes aligns with previously reported data by Chau et al. [13].

Although AMR was not significantly associated with prognosis at the time of analysis, a trend toward significance was noted ($p = 0.073$), highlighting the need to expand the study cohort and extend patient follow-up.

Our findings underscore the importance of measuring anti-HLA antibody levels in patients with HT dysfunction, as these antibodies have a demonstrable impact on the risk of post-transplant complications and negatively affect recipient outcomes. Assessing anti-HLA levels in this patient population allows for individualized selection of optimal therapeutic strategies, which can lead to improved clinical results.

Future studies should incorporate anti-HLA levels into the classification of AMR to guide treatment selection more effectively, in line with recommendations by

Goldberg et al. [15]. Continued research is also needed to refine treatment regimens for transplant dysfunction and to develop measures for its prevention and early detection.

CONCLUSION

The study demonstrates a strong correlation between the presence of circulating anti-HLA antibodies and the development of severe HT dysfunction with an increased risk of adverse events. Early detection of anti-HLA antibodies enables early intervention, helps guide the selection of optimal treatment strategies, and contributes to improved long-term outcomes for heart transplant recipients.

The authors declare no conflict of interest.

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