**ABSTRACT**

**Background:** In recent years mechanical circulatory assist devices became an established option in bridging patients with refractory heart failure to heart transplantation. One of the alleged limitations of mechanical devices is a high degree of antibody production with possible deleterious effect on subsequent heart transplantation outcome.

**Aim:** The main goal of this study is to assess the role of antibodies on the outcome of surgical treatment of patients with end-stage heart failure.

**Method:** Firstly, we present a literature review on the current state of knowledge of possible immunologic mechanisms involved in antibody production in left ventricular assist device (LVAD) recipients, new methods of antibody detection, desensitization strategies and overview of published evidence assessing the impact of sensitization on post-transplantation outcome. In the experimental part of our study we prospectively evaluated the presence of anti-Angiotensin II Type 1 Receptor (AT1R) antibodies in 83 Heart Mate II (HMII) recipients who were implanted at our institution between 2008 and 2012 and survived the first 60 days. On-device survival and device malfunction, major infection, major bleeding and neurologic dysfunction were compared between antibody positive and antibody negative recipients. Out of a total of 83 patients, 69 eventually underwent heart transplantation between October 2008 and August 2014. Overall survival and post-transplant rejection free survival were compared between both groups.

**Results:** Anti-AT1R antibodies were observed in 13/83 (16%) of the recipients before HM II implantation (AT1R+). Four of these patients (6%) were also sensitized against HLA antigens. During the support, 50 patients (71%) who were initially anti-AT1R negative became positive (AT1R-/+) and 20 (29%) remained negative (AT1R-). Two year on – device survival was 78 ± 12% in AT1R-, 60 ± 23% in AT1R+ and 92 ± 6% in AT1R-/+ group (p = 0.409). Freedom from device malfunction, major infection, major bleeding and neurologic dysfunction at two years for AT1R-, AT1R+ and AT1R-/+ was 49 ± 14%, 53 ± 16% and 41 ± 11% (p = 0.875). One and five year post transplant in AT1R- was 88 ± 8% and 76 ± 10% and in 87 ± 5% and 81 ± 7% in AT1R+ (p = 0.582). Freedom from ACR at one year was 68 ± 12% for AT1R- and 75 ± 6% for AT1R+ (p=0.218). Freedom from pAMR was 100% in AT1R- and 98 ± 2% in AT1R+ (p = 0.198).

**Conclusions:** There was no difference in the post HeartMate II implantation survival among patients who were anti-AT1R antibody positive before device implantation and patients who either became positive or remained negative during the support. The incidence of device malfunction, bleeding, infection and neurological dysfunction was not influenced by the presence of anti-AT1R antibodies. Our data also showed no impact of pre-transplant sensitization against HLA antigens on post-transplant survival.