compatible donor. No donor specific anti-HLA class I or class II antibodies were detected prior to transplant using flow cytometry microbead assays and Flow cytometry T and B cell crossmatches. Her initial postoperative course was uneventful. However, on postoperative day five, she was found unresponsive, pulseless, and expired despite extensive resuscitative measures. An autopsy revealed left ventricular subendocardial and intramyocardial hemorrhage with diffuse lymphocytic infiltrates and myocyte damage, consistent with ISHLT Grade 4 rejection. Immunohistochemistry demonstrated CD57+ cytotoxic lymphocytes, consistent with a predominant NK cell population. Recipient genotyping of the killer IgG receptor (KIR) revealed the presence of activating KIR (2DS2, and 2DS3 of KIR B haplotypes), and 2DS4 with its cognate HLA-C1 and C2 ligands in the donor.

**Summary:** This is the first case report of a fatal, accelerated rejection occurring in the context of a predominantly natural killer cell infiltrate in a transplant recipient following a peripartum cardiomyopathy. Since activating KIRs can decrease the influence of the ‘strong’ inhibitory forms of KIR carried by a Haplotypes, this case suggests that the interaction of patient’s activating KIR with donor HLA-C1/C2 ligands may have contributed to NK cell activation and allograft rejection. In support of this hypothesis, recent studies indicated that KIR2DS4/HLA-C ligands increase the risk of early acute rejection in liver transplant recipients. Several mechanisms exist by which NK cell activation may have led to rejection in this case, including ischemia time, viral pathogens, and KIR/HLA class I ligands interaction.

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**Autoantibodies Against Lung Tissue Can Cause Hyper Acute as Well as Acute Antibody Mediated Rejection Following Lung Transplantation**

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**Introduction:** Autoantibodies (AAbs) against lung antigens collagen type I (ColI), type V (ColV), and k-anti tubulin (KAT) predispose to chronic rejection following lung transplant. Here we demonstrate for the first time that pre-existing AAbs can lead to hyperacute rejection while de novo AAbs can cause acute antibody mediated rejection (AMR).

**Case Report:** A 63-yo old female with emphysema underwent single right lung transplant without cardiopulmonary bypass (CPB). Pulmonary pressure was normal and she had no risk factors for primary graft dysfunction. Cold ischemia was under 3 hours and implantation was uneventful. However, 30 minutes following reperfusion, the allograft became congested and the patient developed increased FiO2 requirement to 100%. Velocities across the arterial and venous anastomoses were normal. Post-operative chest radiograph (CXR) showed complete opacification of the transplanted lung. HLA panel reactive antibodies (PRA) and the prospective as well as retrospective cross-matches were negative. Lung biopsy was consistent with AMR and showed both complement C4D and IgG deposition. Retrospectively tested, pre-transplant sera demonstrated high CoI and CoV AAbs with moderate KAT Abs titers. Donor and recipient airway cultures did not show any growth. Antibody directed therapy (ADT) including IVIG resulted in prompt improvement of the transplanted lung and the patient was discharged breathing room air. The left lung from the same donor was transplanted at a different center into a 66-yo male with IPE and pulmonary hypertension using CPB. The post-transplant course was uneventful. However, he was readmitted at 3 weeks from transplant with respiratory distress and opacification of the left lung allograft. Lung biopsies indicated AMR despite negative PRA and cross-match. Pre-transplant AAbs were found to be negative but he had developed de novo AAbs against all three antigens just prior to AMR. ADT lead to resolution of the AMR and clearing of the CXR. The patient was discharged requiring 1L/min supplemental oxygen only on extubation.

**Summary:** AAbs can lead to both hyperacute and acute rejection of lung allograft. Prompt recognition and treatment of AAb mediated-rejection can salvage the allograft and prevent need for retransplant. We propose that prospective screening of AAbs should be performed in lung recipients.

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**Successful Treatment of Severe Acute Graft Versus Host Disease Post Lung Transplantation**

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**Introduction:** Acute graft versus host disease (GVHD) is an exceedingly rare and fatal complication of lung transplant (Ltx). We report a case of acute GVHD post Ltx presenting with severe persistent pancytopenia, abnormal liver enzymes and skin rashes. The diagnosis was made by peripheral blood short tandem repeat (STR) assay and skin biopsy. Although unresponsive to therapy with high dose steroid, he was successfully treated with anti-thymocyte globulin (ATG).

**Case Report:** A 48-year-old male with history of sarcoidosis status post bilateral Ltx presented with fatigue, severe leukopenia (WBC 0.2 thou/cu) and abnormal liver enzymes 12 weeks post transplant. The post-transplant course was uneventful with no acute rejection or infection. His immunosuppression included tacrolimus, mycophenolate, and prednisone. Despite withholding mycophenolate, valganciclovir, TMP/Sulfa, voriconazole and initiation of daily G-CSF, patient progressed to pancytopenia, worsening liver enzymes and new maculopapular rashes. This prompted a bone marrow biopsy showing an aplastic bone marrow. Infectious work up was unrevealing. Subsequent peripheral blood STR showed engraftment of donor cells with 99% of T cells and 65% of B cells were from the donor origin. The diagnosis of GVHD (grade 3) was made based on STR result, and consistent skin biopsy findings. The initial therapy with high dose steroids was unsuccessful with no bone marrow response and worsening liver enzymes. This led to a four-day trial of therapy with ATG. A repeated peripheral STR 5 days post ATG showed resolution of macrochimerism with resolution of pancytopenia.

**Summary:** Only 10 cases of GVHD post Ltx have been reported with an 80% mortality. The two patients who recovered had milder form of GVHD and responded to high dose steroid. We report a case of severe GVHD refractory to steroids and later successfully treated with ATG. Our patient had 1/6 HLA conformity with the donor, unlike the suggested high degree of conformity as a risk factor for GVHD. In the absence of any established risk factors, GVHD should be suspected in the presence of persistent pancytopenia, skin rashes and abnormal LFTs of unclear etiology post Ltx. Additionally, strict infection precautions during the protracted neutropenia while identifying the appropriate therapy are crucial for successful therapy.

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**Breaking Bad: Dissimulated Amphetamine Abuse as a Rare Cause of Recurrent LVAD Pump Thrombosis**


**Introduction:** Recent reports about accumulating incidence of LVAD pump thrombosis occurring in continuous-flow pumps emphasize the relevance of this major complication.

**Case Report:** We report here a 44 year old male patient with his third pump thrombosis after HVAD implantation with overall well-adjusted coagulation status using phenprocoumon (INR 2.0-2.5) and ASA/dipyridamol. In February 2013 LVAD implantation was performed due to cardiogenic shock as a result of a non-ischemic dilated cardiomyopathy. The post-operative recovery proceeded without complications. The patient presented with LVAD alarms due to power consumption elevations 6 month after LVAD implantation. LDH levels were 4122U/l. Therefore an intravenous thrombolysis was performed in order to resolve the thrombus. After normalization of clinical and laboratory values, a malposition of the inflow cannula was excluded by chest X-ray. A second anti-platelet agent (dipyridamole) was added to the anti-thrombotic therapy. 4 month later the patient presented again with LVAD alarms due to power elevations. LDH levels were elevated (3497U/l) and therefore thrombolysis was administrated intravenously again. Outpatient follow-ups were completely uneventful with normal LDH levels and INR values in range (INR 2.0-2.5). 14 months after LVAD implantation the patient developed a pump thrombosis again (LDH 5845U/l). Thrombolysis was performed to resolve the thrombus once again. Analysis of the coagulation status did not reveal crucial explanation of the recurrent pump thrombosis. Even though drug abuse was denied by the patient, a drug screening test was performed. Positive amphetamine and metamphetamine findings were detected and explain the recurrent pump thrombosis. Amphetamines are known to be