**Acute Decompensation in a Heart Transplant Patient**

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**Abstract:**

**Background:** After heart transplantation, opportunistic infections are the leading cause of death between six months to one year post-transplant. Cytomegalovirus is a frequent complication in post-transplant patients, and invasive disease can manifest cytopathic effect in various organs. Ureaplasma has been mostly reported as a cause of severe hyperammonemia in lung transplant patients, and of pneumonitis in immunocompromised children. Parvovirus B19 can cause significant morbidity including hematologic abnormalities, myocarditis, and allograft dysfunction.

**Case Description:** We report the case of a 16 year old female who underwent heart transplant for dilated cardiomyopathy. Donor and recipient were both positive for CMV titers, and she was placed on valganciclovir for 6 months. 9 months post-transplant she presented with back pain, abdominal pain and emesis and began treatment for rising CMV titers (971 IU/mL) and presumed rejection. On hospital day 3 she had an acute decompensation requiring cannulation onto VA ECMO, and cardiac function decreased from EF 47% to less than 10%. Biopsy revealed mild antibody-mediated rejection grade pAMR 1 (H+), and focal myocyte injury with very minimal lymphocytic infiltrate. HLA antibody titers were fairly unremarkable. Extensive workup was significant for positive Ureaplasma parvum blood PCR, and accompanying hyperammonemia to 400 which was treated and resolved. Despite aggressive treatment for CMV and Ureaplasma, she continued to deteriorate with multi-organ failure of the liver, kidneys, and extensive necrosis of lungs. Repeat cardiac biopsies revealed extensive myocyte necrosis and she became asystolic on hospital day 20. After discussion with family, mechanical support was withdrawn after 8 weeks. Preliminary autopsy findings revealed parvovirus B19 on cardiac specimens, in the context of negative blood PCRs.

**Conclusion:** This case illustrates an acute decompensation requiring emergent mechanical support after systemic CMV and Ureaplasma infection, as well as myocardial parvovirus B19 infection. The cardiac allograft dysfunction was likely due to the combination of some or all of the infectious etiologies resulting in myocardial injury, but it is difficult to determine the cause with certainty.