Acute respiratory distress syndrome after heart transplantation in a highly sensitized recipient: a case report

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Introduction: Human leukocyte antigen (HLA) sensitization limits donor availability and increases the risk of waitlist mortality, antibody-mediated rejection (AMR), cardiac allograft vasculopathy, and reduced survival after heart transplantation (HTx). Management often requires complex crossmatch strategies and intensified immunosuppression such as rATG, IVIG, plasmapheresis, rituximab, or complement inhibitors, which may increase complications.[1-3]

Case report: 21-year-old woman with arrhythmogenic right ventricular cardiomyopathy and high sensitization (cPRA 70%) underwent orthotopic HTx. She received immunoadsorption, rATG, corticosteroids, IVIG, and maintenance with tacrolimus and mycophenolate. On POD 4 she developed dyspnea with bilateral infiltrates, progressing to ARDS by week 2 and requiring mechanical ventilation. Lung CT showed diffuse ground-glass opacities and consolidation. Infection was excluded and graft func-

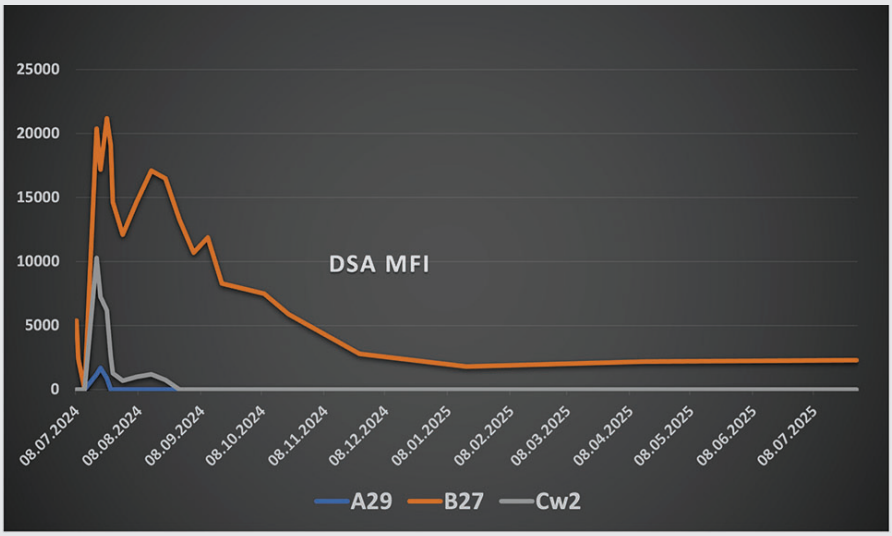

FIGURE 1. The patient's clinical course with treatment.
***Luminex before the 5th immunoadsorption, ** Corticosteroid dose was intravenous methylprednisolone 125mg for 5 days.**
A29, B27, Cw2- Human Leukocyte Antigens, MFI- mean fluorescence intensity, Bx- endomyocardial biopsy, IVIG- intravenous immunoglobulin, IgM- immunoglobulin M, IAS- immunoadsorption, CTS- corticosteroids.

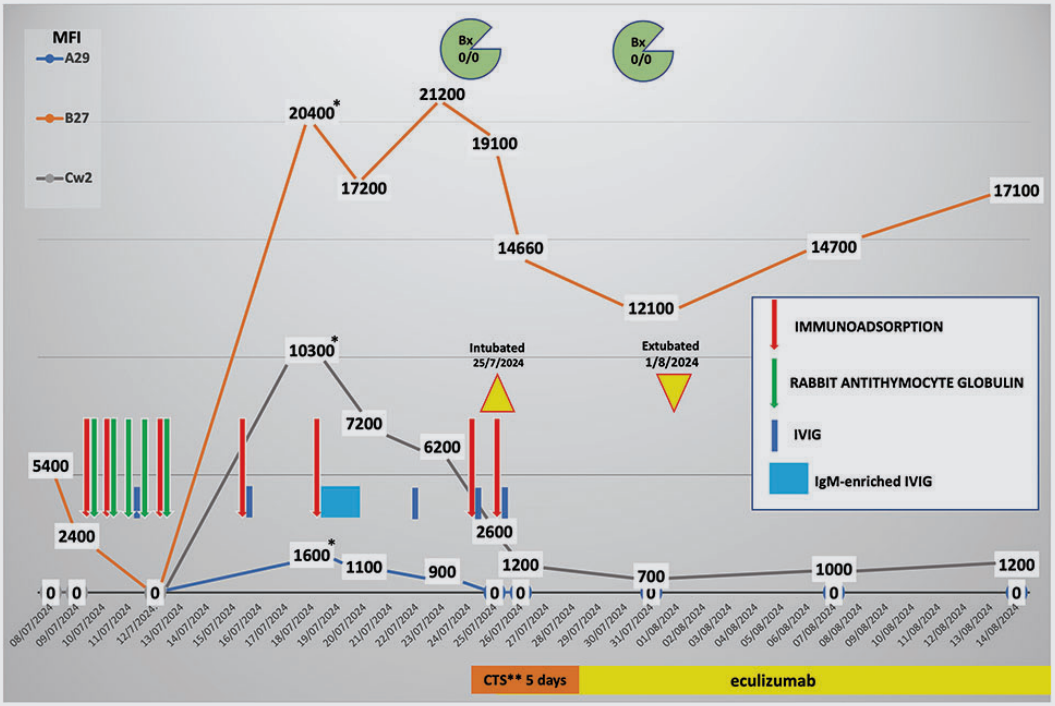

FIGURE 2. Temporal evolution of Donor Specific Antibodies (DSA) Mean Fluorescence Intensity (MFI).

tion remained normal. Despite treatment, donor-specific antibodies rose (A3 900 MFI, B27 21,200 MFI, Cw2 6,200 MFI). Lung injury was suspected from IVIG or rATG. Further IAS/IVIG were withheld, and eculizumab introduced. Corticosteroid pulses were given for ARDS. She was extubated after 7 days with rapid clinical recovery, though infiltrates persisted for 4 weeks (**Figure 1**). At 12 months she was asymptomatic, rejection-free, and had low DSA (B27 2,300 MFI) (**Figure 2**).

Discussion: Pulmonary toxicity after IVIG or rATG is rare. IVIG-related lung injury is usually reversible, including pneumonitis or diffuse alveolar damage via hypersensitivity or immune-complex deposition. rATG more often causes severe complications such as ARDS, often during first infusions, through cytokine release or TRALI-like reactions involving complement activation, direct toxicity, or hypersensitivity. Eculizumab has not been associated with acute lung injury and may mitigate complement-mediated endothelial injury and capillary leak.

Conclusion: In highly sensitized HTx recipients, complex immunotherapy entails substantial risk. Flexibility in therapeutic strategies is essential to reduce the high risk of rejection and graft dysfunction. In this case, eculizumab may have contributed not only to rejection prevention but also to pulmonary recovery.

LITERATURE

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