Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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# Eculizumab therapy in the late onset atypical Hemolytic Uremic Syndrome in a kidney transplant recipient : case report

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**Case Study :** A 52-year-old male presented with sudden decrease of kidney function. He underwent ABO-incompatible living donor kidney transplantation for IgA nephropathy, 10 years ago. His post-transplant course had been stable for eight years (sCr 1.03 mg/dL) until the eighth year, when chronic active antibody-mediated rejection (cAAMR) and recurrent IgA nephropathy caused his baseline sCr to shift to 2.0 mg/dL. Two weeks prior to the current admission, he contracted a norovirus infection. Despite clinical recovery from the virus, his sCr abruptly rose to 8.99 mg/dL. Laboratory findings indicated thrombotic microangiopathy (TMA), showing microangiopathic hemolytic anemia (Hb 7.6 g/dL, LDH 629 U/L, low haptoglobin) and thrombocytopenia. Allograft biopsy confirmed definitive TMA features, including acute and chronic microcirculation injury with severe interstitial fibrosis. Notably, C4d staining was negative and donor-specific antibodies (DSA) were absent, making ABMR an unlikely cause for the acute spike. Further workup revealed normal ADAMTS13 activity (71.2%) and negative Shiga toxin tests. A genetic panel identified a heterozygous missense variant in the complement factor H (CFH) gene (p.Gly1110Ala), which in-silico tools predicted to be deleterious, supporting a diagnosis of atypical hemolytic uremic syndrome (aHUS). Management began with plasmapheresis and corticosteroids, but severe allergic reactions necessitated a switch to eculizumab. The patient received 900 mg weekly for four weeks, followed by 1,200 mg every two weeks. Following treatment, the patient achieved complete hematologic remission; haptoglobin levels normalized to 213.9 mg/dL and LDH returned to baseline. However, due to the severity of the initial insult and underlying chronic damage, renal function did not recover, leaving the patient dialysis-dependent. This case highlights that late-onset aHUS can occur even years after transplantation. When conventional therapies like plasmapheresis are not tolerated, eculizumab is vital for controlling systemic complement activation and ensuring patient survival.

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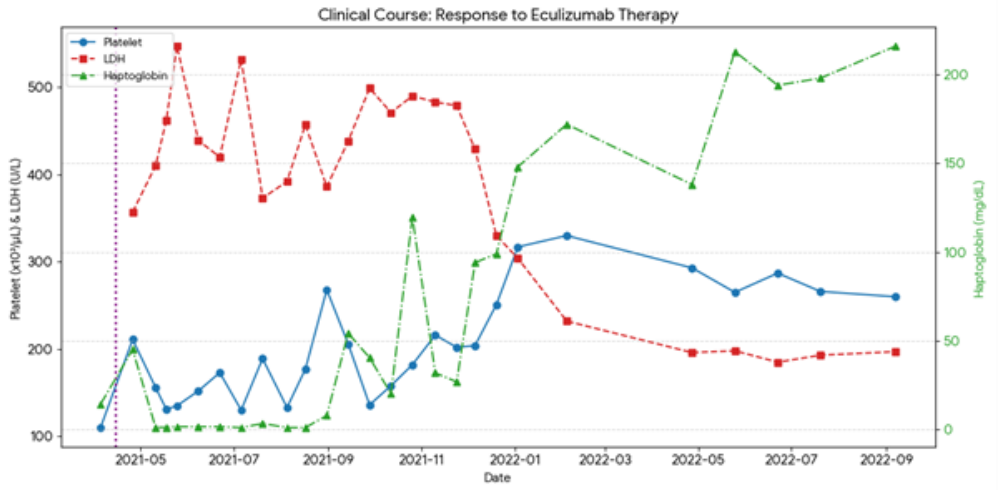
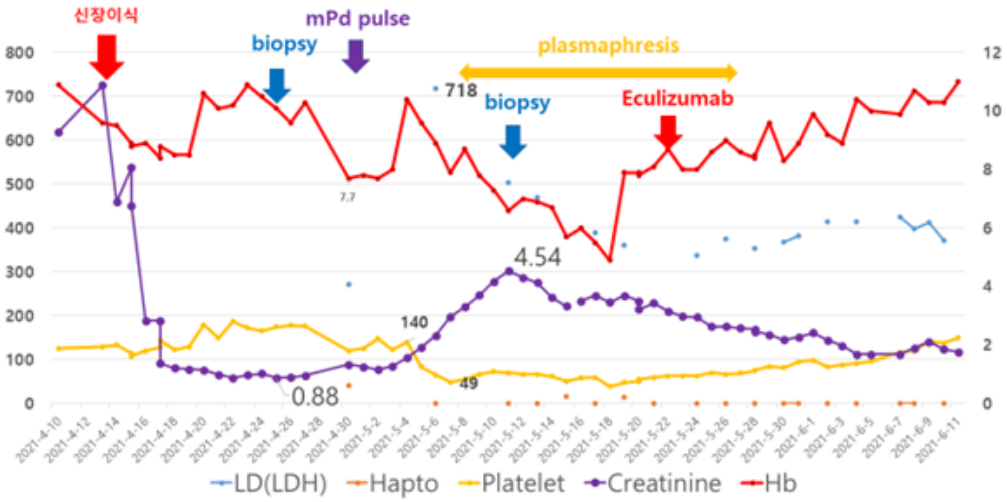

Clinical Course: Response to Eculizumab Therapy
Platelet
LDH
Haptoglobin
Platelet (x10^9/μL) & LDH (U/L)
Haptoglobin (mg/dL)
Date
2021-05
2021-07
2021-09
2021-11
2022-01
2022-03
2022-05
2022-07
2022-09

그림 1.png


신장이식
mPd pulse
biopsy
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718
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Eculizumab
7.7
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Hapto
Platelet
Creatinine
Hb